Official Transcript of Proceedings ACNW T-0175

NUCLEAR REGULATORY COMMISSION

Title: Advisory Committee on Nuclear Waste
174th Meeting

Docket Number: (not applicable)

Location: Rockville, Maryland

Date: Wednesday, November 15, 2006

Work Order No.: NRC-1322 Pages 1-235

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UNITED STATES NUCLEAR REGULATORY COMMISSION'S

ADVISORY COMMITTEE ON NUCLEAR WASTE

November 15, 2006

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UNITED STATES OF AMERICA

NUCLEAR REGULATORY COMMISSION

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ADVISORY COMMITTEE ON NUCLEAR WASTE (ACNW)

174RD MEETING

THIRD DAY

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WEDNESDAY,

NOVEMBER 15, 2006

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The meeting was convened in Room T-2B3 of Two White Flint North, 11545 Rockville Pike, Rockville, Maryland, at 8:30 a.m., Dr. Michael T. Ryan, Chairman, presiding.

MEMBERS PRESENT:

MICHAEL T. RYAN	Chair
ALLEN G. CROFF	Vice Chair
JAMES H. CLARKE	Member
WILLIAM J. HINZE	Member
RUTH F. WEINER	Member

ACNW STAFF PRESENT:

JOHN T. LARKINS, Executive Director, ACRS/ACNW

LATIF HAMDAN

ANTONIO DIAS

DEREK WIDMAYER

JOHN FLACK

RATEB M. "BOBBY" ABU-EID

ALSO PRESENT:

THEODORE ROCKWELL

BERNARD LE GUEN

YVES GARCIET

DAVID KOCHER

JIM MUCKERHEIDE

ALEXANDER WILLIAMS

GLENN REEVES

RAY WYMER

LARRY TAVLARIDES

HARRY LARSON

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Adjourn	

P R O C E E D I N G S

8:33 A.M.

CHAIRMAN RYAN: The meeting will come to order, please.

This is the third day of the 174th meeting of the Advisory Committee on Nuclear Waste. During today's meeting, the Committee will consider the following: dose effect relationships and estimation of the carcinogenic effects of low doses of radiation radiation; a white paper on potential advanced fuel cycles; and discussion of ACNW draft letter reports.

This meeting is being conducted in accordance with the provisions of the Federal Advisory Committee Act. Latif Hamdan is the Designated Federal Official for this meeting. There he is, Latif, thank you.

We have received a request by Dr. Theodore Rockwell from Radiation Science and Health, Incorporated to make an oral statement during today's session and we'll schedule that. We'll get that organized for a presentation in a short while. Should anyone else wish to address the Committee, please make your wishes known to one of the Committee staff.

It is requested that speakers use one of the microphones, identify themselves and speak with

sufficient clarity and volume so that they can be readily heard.

It's also requested that if you have cell phones and pagers that you kindly turn them off.

Thank you.

(Pause.)

I'm pleased to tell you this morning that we have members from the French Academy of Science Committee on the Dose Effect Relationships and Estimation to Carcinogenic Effects of Low Doses of Ionizing Radiation Report recently published by the French Academy. Our presenter is Dr. Bernard Le Guen and accompanying him is Dr. Yves Garciet, also involved with radiation protection in France.

Gentlemen, welcome to the United States and welcome to the ACNW and we truly appreciate your willingness to come and share this presentation of your report and without further delay, I will turn the presentation over to Dr. Le Guen. Welcome and thank you.

DR. LE GUEN: Thank you. So good morning, ladies and gentlemen. I would like to thank the organizer for the invitation. I'm Dr. Le Guen. I'm a medical advisor at EDF and I'm also the president of Health and Research section of the French Radiation

Protection Society.

I am also a co-author of the French Academie report.

Over the past 20 years, the French Ministry of Research has twice asked the Academie des Sciences to carry out the critical review of the available data regarding the effects of low doses of ionizing radiation on health.

In 2003, the two Academies, Academy of Science and the National Academy of Medicine, decided to join their effort for an update of two main topics: the dose-carcinogenic effect relationship and the carcinogenic effect of low doses.

A working party was set up; about 50, 52 different versions and its report was accepted after a few modifications, suggested by the reviewers and it was released in March 2005.

The main problem for both medical and nonmedical uses of ionizing radiation is the possible carcinogenic risk associated with small doses of ionizing radiation. These eventual risks are also of great importance with regard to natural irradiation. Just an example: it would be of great value to assess the risk of lung cancers caused by various radon concentrations in the air at home or at work, and

whether there is a practical threshold below which the risk becomes negligible.

And in our estimation of the risk associated with exposure to radon at home, could lead either to overlooking serious public health problems given the number of people exposed or conversely, to ensuring considerable pointless expense in order to limit such exposure.

The assessment of carcinogenic risk associated with doses of ionizing radiation from 0.2 sieverts to 5 sieverts is based on numerous epidemiological data. However, the doses which are delivered during medical x-ray examination or the doses received by nuclear workers or in regions of high natural background irradiation are much lower from 0.1 millisieverts to 20 millisieverts.

The evolution of the cancer risk of low doses is of great importance in medicine. Just an example about France, approximately 17 million radiological examinations are performed in France every year, delivering an average of 1 millisievert per year to every French person. Depending on the dose effect relationships used, it can be deduced from this either that these exams could be leading to about 3,000 cases of cancer a year or that they do not

represent any hazard.

To illustrate my point, doses delivered during a medical x-ray examination and you can see that it depends which exam is performed, so it's from 15 microsieverts in case of chest x-ray to 4 or 10 millisieverts in case of body scan.

In fact, in Europe, there is a large discrepancy, a large variability in the dose received for the same examination from one country to another. From my point of view, before to assess precisely the risk the first step for us is a step of optimization, is a step of harmonization of the common practice in Europe because you can see that it's not the dose, it's the skin dose, milliGray. If you have a chest x-ray in the Netherlands, you will receive 0.13 milligray, but in Greece, you will receive 1.93 milligray.

About now as the dose received by nuclear workers and by population who live in the vicinity of nuclear power plants, nuclear energy delivers about 0.001 millisieverts so one microsievert per year to each performed in France in the vicinity of four plants, the dose can reach 15 millisieverts, 15 microsieverts, sorry, 15 microsieverts per year. So people working in the nuclear industry receive on

average less than 2 millisieverts per year.

And you can observe the large reduction of 50 persons over the last 10 years with to date an average dose of 1.6 millisieverts per year, so very close to the natural background.

So the impact on health varies widely depending on how it is estimated between zero impact and several dozen lung cancers per year for the entire French population and between zero in a few lung cancers per year for workers.

Here is the same diagram that's concerning the collective dose with a large decrease of the collective dose over the last 20 years for the same number of reactors in France and today, the collective dose is about 0.78 Man.sieverts.

Well, following the small doses, no excess of cancers has been detected. However, the lack of an increase does not exclude possibility of a small excess of cancers. Solid tumors and leukemia have a spontaneous incidence that is high and varies according to lifestyle. Just an example here, due to the aging process, you have the increase of the incidence of the breast and colon cancer and those without exposure to ionizing radiation, just due to the aging process.

So, the possible increase in this incidence following irradiation is relatively low, so the studies must have sufficient statistical power which require large cohorts. But, in large populations, confounding factors as consumption of tobacco, for example, are present and they must be taken into account by appropriate statistical methods because their specific effect can be much greater than the effect of irradiation.

So, it is highly unlikely that putative carcinogenic risk could be estimated in the future or even established for low doses through case control studies or the follow-up of cohorts due to the all-confounding factors.

Well, both of the difficulties about epidemiological studies, you know this, that's if you have a high dose with a dose received about one sievert, one thousand millisieverts, you need a cohort, you know, an epidemiological, one moment, an epidemiological study of 500 people and conversely, if you have a low dose, about ten millisieverts, you need five million people in your cohort.

Other confounding factors are the natural irradiation background. You need to take into account the cosmic radiation, you know that it's different if

you live at sea level or if you live in altitude. You need to take into account the external exposure to earth's radiation. Of course, you know the famous example about Brazil, that's for Antonio, I don't know where it is. The sun, you have 35 millisieverts per year. And you have also internal exposure due to drinking water. I gave just an example with the French St. Alban water, and you can receive 1.25 millisieverts per year.

So, even for several hundreds of thousands of subjects, the power of such epidemiological studies would not be sufficient to demonstrate the existence of a very small excess in cancer incidence or mortality aiding to the natural cancer incidence which in a nonirradiated population, is already very high and fluctuates according to lifestyle. And, today, because of these epidemiological limitations, the only method with epidemiological studies for estimating the possible risk of low doses, so below 100 millisieverts is extrapolation from carcinogenic effects observed between 0.2 and 3 sieverts, with all the friction exposed.

Well, the French reports point out that following exposure to low doses, epidemiological studies have not evidenced any significant effect

because either there is no effect or the effect is too small to be detected by such studies. These results, which are sometimes described as negative results are useful because they help to assess the upper limit of the potential risk and can be included in meta-analysis.

I would like to give you some examples. Of course, you know the famous cohort of Hiroshima/Nagasaki. We have 76,000 people in the cohort with an average dose of 200 millisieverts. There is no risk, the risk is not significant for leukemia below 150 millisieverts and the risk is not significant for solid cancer below 100 millisieverts.

The first ICRC publication in 1995 with three cohorts, with three countries, when you have 96,000 nuclear workers, the risk was not significant for leukemia below 400 millisieverts. They observed an increase of the risk of leukemia in the first study upwards of 400 millisieverts and for solid cancer, it was not significant.

The last ICRC publication, published in 2005, with a large cohort, 600,000 nuclear workers, with a small dose received, an average of 19.4 millisieverts. The leukemia and solid cancer was not significant below 100 millisieverts, but they conclude

that one or two percent of the cancer observed are due to ionizing radiation. But there is much larger, much increase of the uncertainty in this publication than the other one.

And you can see that you have a large cohort. It's not because you have a large cohort that you have not uncertainties. I will give you just an example in a few moments about that.

Other publications, radiologists, about exposure from 1960 to today, a large group of physicians, 220,000 physicians in this group, the dose received from 10 to 15 millisieverts per year, and the risk of leukemia and solid cancer were not significant.

Cabin crew, a group of 47,000 people with a low dose exposure from 1.5 to 6 millisieverts per year, the leukemia and solid cancer were not significant, but they observed an increase of melanoma. And you must, perhaps you know that melanoma is not related to ionizing radiation exposure. The increase is probably due to long exposure to the sun, to UV, probably on the beach during the different stop-overs, but not due to the ionizing radiation exposure.

Well, another example is about medical

examination. There is an interesting American publication on the women exposed by fluoroscopy. It was due to tuberculosis disease and the fluoroscopy was used just after World War II so from 1945 to 1960, and these were large cohorts and they observe an increase of breast cancer for an exposure of about 100 millisieverts.

About radiotherapy, another publication with 7,700 breast cancer, the excess of solid cancer was not significant. For the tissue, while the dose received was below 150 millisieverts. So, not on the tumor, but on the border, on the tissue borders the tumor, when you cannot receive some exposure and the risk was not significant below 150 millisieverts.

Now, some important studies, some important new facts have emerged, such as the feasibility and value of studies comparing the morbidity and mortality in regions with high and low levels of natural irradiation, but similar lifestyles. And, for the moment, for example in Kerala in India with a publication with 100,000 people with a dose, an average dose of 70 millisieverts per year, there is no increase in incidence of leukemia or solid cancer.

Another publication in China, in Yangijang, with low exposure from 2 to 6 millisieverts

per year, and the risk for the moment was not significant or for us, the publication, so it's no risk of leukemia or solid cancer.

Well, about the last slide here, see publication, here you have different cohorts. You have the Canada cohort, the Sweden, U.K. and Germany, American cohort and when you combine all the cohorts, you observe an excess relative risk per sievert for all concerned excluding leukemia in cohort of more than 100 deaths. If you have a look, for each cohort, the risk was not significant except for Canada's cohort and if you don't take into account Canada's cohort, the risk is not significant. So there is a problem of heterogeneity in this cohort with this group.

Another problem, another difficulty was the typical consumption of the lung cancer. They weren't able to take into account as a typical consumption and see, if you don't take into account the lung cancer, there is no risk, so don't observe an excess of risk. So it's very hard to conclude and in fact, you can see that this second ICRC publication of much more uncertainties than the first study published in 1995 with less workers included in the cohort.

Well, so the question remains, here you have the recognized effect, so the question is what is the good relation between dose and effect below those recognized effect? Is it linear relationship? Is it a quadratic relationship? Or is it a normal relationship?

In fact, the relationship takes into account the linear no threshold is not a problem for regulation, but the question is, is it true or is it not true?

Well, a few comments about linear no threshold relationships. The LNT model was used in 1966 by Russell to evaluate the radio-induced mutations in the germ cell line in the mouth. It was introduced between 1960 and 1980 for the purposes of regulation in radiation protection with regard to all mutagenic and carcinogenic effects in humans.

At that time, LNT was considering a convenient and pragmatic relationship, but a model based on scientific data. In the 1960s, the International Commission of Radiation Protection introduced it because it allows the addition of sequential irradiation delivering or low or high doses of radiation received by an individual whatever is the dose rate and the fractionation.

Thus, it greatly simplified accounting in the radiation protection, however, gradually LNT was interpreted as meaning that the carcinogenic risk is proportional to the dose and that even the smallest dose induces a cancer risk.

So because we think that epidemiological studies do not have sufficient statistical power to determine the risk from low-dose exposures, therefore fundamental mechanistic studies are essential to understand biology short and long-term effect of low-dose ionizing radiation and to help evaluating risk at those dose levels.

Recent research developments and in particular, molecular approaches have lead to new findings that put into question some of previously established radiobiological paradigms and concepts.

The present review outlines what we got to know recently. What we'd still like to know of low dose and low dose rate effects and the possible consequences for radiation protection.

Well, the rapidly growing knowledge in molecular biology and radiobiology during the last decade should let us to examine the validity of the implicit assumption on which the use of LNT has been based for assessing the carcinogenic effect of low

doses below 100 millisievert and of very low doses before 10 millisievert on the basis of that observed in the range of doses of 0.2 to 3 sieverts.

The LNT model postulates that the cell reacts in the same way regardless of the dose rate and dose which implies that the probabilities of death and mutation, their unit dose and the contribution to carcinogenesis of each physical event remains constant, irrespective of the number of lesions in the cell and in the neighboring cells.

This constancy implicitly admits several hypotheses. First, in the range of the doses and dose rates and their consideration, there is no physical, chemical or biological interaction between the effects caused by the various particles in the cell and we know that is not true.

Second, any absorbed dose of energy in the cell nucleus leads to a proportional probability of mutation and we try to show to you that is not true.

Third, the probability of successful repair of misrepair per dose unit are always the same whatever the number of lesions of the same cell. That is not true.

Fourth, there should be no intact of dose or dose or those rates. Similarly, the probability of

a part of this does not vary with those. It's not true. And last, any DNA lesion has the same probability of giving rise to cancer, irrespective of the number of alterations in the stem cell and in the neighboring cells. We will try to demonstrate that it is not true.

Well, so the LNT has been used for assessing the effect of low-dose and very low doses. This procedure has become a dogma in many radiation protection cycles. But the validity of the LNT has been challenged over the past decade for too many reasons. Some meta-analysis of the animal data have shown the absence of any carcinogenic effect of doses below 100 millisieverts. I put just an example with Phillip Duport meta-analysis, with more than 60,000 mouses on the anomalies effect with 40 person of the experimenter series.

And scientific progress, and I will talk about scientific progress. Scientific progress has revealed the complexity of carcinogenesis and the diversity and effectiveness of the responses of a cell to radiation. So this LNT hypotheses are not consistent with current radiobiologic knowledge which shows that cells do not remain passive when they are irradiated, either by solar UV or by radiation.

Because of course, ionizing irradiation is not the only genotoxic for the cell.

Moreover, intracellular communication systems inform a cell about the presence of an insert in neighboring cells. Of course you know in the case of ionizing radiation, DNA is a target. And the question is, is there a probability of DNA mutation or not?

The oxidative stress induced by irradiation triggers several defense mechanisms against detoxify active spaces. Directive oxygen spaces formed by water induced by radiation damages some cell constituent and produces oxidative stress.

This oxidative stress stimulates enzyme systems that detoxify active spaces of oxygen formed and induce the synthesis of enzymes that destroys them. In parallel, oxidative stress also activates numerous signaling pathways. In case of DNA damage, it's not the in cell physical, chemical event that changes, but their outcome.

This sentence is very important. The defense mechanisms induced in a cell depends on the degree and the nature of the cellular damage. So in the case of low linear energy transfer, so LETs, so in the case of low linear energy transfer radiation, such

as photons or electrons, when the warm body is exposed to one milligray, each cell is on average crossed by one electron.

Each electron induces an average of two DNA lesions, including one single-strand break, one SSB, and four by ten to the minus two double-strand breaks, DSB, of the DNA molecule. And ten to minus four chromosome aberrations. This initial effect is proportional to the dose. As in general, DSB is a direct or in direct consequence of high transfer of energy within or alongside DNA molecule, mainly by means of radiation induced active oxygen spaces.

The defense mechanism, induced in the cell depends on the number and nature of cellular damages. The number of double-strand breaks caused by one gray dose has been estimated to be between thirty and forty. In contrast, the number of double-strand breaks of endogenous of natural origin of the stress produced in each cell by the oxygen's metabolism remains controversial.

It has been estimated to be eight per day and 50 per cell cycle, by (9:03:39), who estimates that about one person's single-strand breaks turn into double-strand breaks, and it assumes 3,000 single-strand breaks per day in a cell. So we tried to

resume now differences between endogenously and ionizing radiation induced DNA lesions endogenously due to cellular metabolism, one finds many single-strand breaks and modified bases.

However, also double-strand breaks are complex lesions. Ionizing radiation induced lesions in DNA include considerable amounts of double-strand breaks and complex cluster of lesions such as locally multiply damaged sites, LMDS, together with many single-strand breaks and base damages.

Well, for example you have here the comparison between endogenous and radiation induced DNA damage. You have here for spontaneous lesion per cell per day and here you have radiation induced lesions per gray.

That's very interesting to note that the double-strand breaks caused by natural irradiation of 2 to 25 millisieverts per year only corresponds to a very small fraction of the total number of double-strand breaks, less than one per thousand. That's normal because ionizing radiation is not the only stress for the cell.

We will talk about clustered damaged, LMDS, because it seems to be specific for ionizing radiation. The first physical chemical events trigger

a series of signals and reactions that can profoundly alter the fate of the DNA lesions. So is this not the initial physical chemical events that change, but their outcome?

The defense mechanism induced in a cell depends on the number or nature of similar damages. Modern transcriptional analysis of cellular genes using micro-array technology reveals that without modification of the genome, numerous genes are activated or innovated following doses much lower than those for which mutagenesis is observed.

Moreover, depending on the dose and the dose rates, not the same genes are transcribed. In the nucleus, different degrees of DNA damage lead to the activation of different family of genes. And now I will show to you a few examples in a few moments.

In recent years, some new findings have alerted radiation biologists. K-shell activation by low LET ionization radiation and the emission of two Auger electrons, 250 and 360 electron volts, can induce complex DNA damages like DNA double-strand breaks. Also, very low energy electrons below 10 electron volt can give rise to double-strand breaks. And high LET and low LET ionic radiation can give rise to locally multiplied damaged sites in DNA.

In the light of theoretical considerations and in vitro and to the only in vitro experimental studies, it has been proposed that ionizing radiation could induce multiple localized lesions consisting of single-strand breaks, oxidative damage to bases, and clusters of double-strand breaks located within a distance of less than 20 base pairs within the DNA.

These very complex lesions are considered to be responsible to a large extent for the genetic effects of radiation. They may constitute particular obstacles to cellular repair.

Well, so predicted from biological, biophysical model calculation, from Monte Carlo calculation, true to be induced at higher levels at low leads radiation, and as I say, they may consider particular obstacles to cellular repair.

In contrast to lesion arising during normal cellular metabolism, clustered lesions or LMDS are thought to constitute molecular markers or signatures of ionizing radiation and to be rather exclusively induced by ionizing radiation, see BEIR VII report.

In addition, 30 percent of double strand rates are of complex form. So LMDS are thought to be responsible for most genotoxic effects such as

lethality, mutations, chromosome aberrations, cell transformation and cancer. This is in the BEIR VII report.

In fact, much work has been done in recent years to better define and quantify these lesions in irradiated cells and determine their biological consequences. You can see publication of Sutherland and Gulstion and Young and in front with Boucher.

So according to BEIR VII, LMDS, clustered damage, may be viewed as complex lesions associated with ionizing radiation and not with endogenous oxidative processes. If there are refractory to repair, the risk of humans posed by ionizing radiation may be viewed as grater than that posed by endogenous oxidative stress.

But in fact, however, in LMDS, today, are difficult to quantify in human cells and their number, if present, is quite limited.

Most of cluster lesions may consist of complex double-strand breaks. In most cases, that's true, plus clustered lesions are found refractory to repair, but such lesions are lethal and nonmutagenic. so if the cell dies, there will be no consequence for the tissue.

The tests are unlikely to contribute

significantly mutagenic and carcinogenic risk of ionizing radiation for humans. So differences about DNA repair, this conclusion regarding differences in the efficacy of the protection system are supported by various experimental or clinical data which highlights the impact of repair and the biologic consequences of the radiation.

So about repair and dose rates, at equal doses, the mutagenic effect varies markedly with the dose rates. When the dose rates increases the mutation frequency after having passed through a minimum increases strongly. A limited number of lesions incudes a reversible arrest of the cell cycle with repair. And conversely, the high local density of lesion reduces the repair efficacy.

So dose rate effects on cell survival and the induction of DSBs in mammalian cells. While the dose rate is low, the number of lesions simultaneously presented in the cell is limited.

Conversely, the high dose rate leads to the simultaneously presence of a large number of lesions. So this high local density of lesions interfere with the coordinated action of the repair system and also increases the probability of error prone enjoining due to the presence of several double-

strand breaks in a restricted volume.

As viewed here, with much more residue or double-strand breaks for the same dose, 11, but in one case that's high dose rates and in the other case a low dose rate.

So at equal doses, the mutagenic effect varies markedly with the dose rate. When the dose rate increases the mutation frequency increases strongly. If the number of lesions which are present simultaneously is small, repair, is generally more effective. Plus, it is more effective at low dose rate than at high dose rate. So in this publication, the introduction of double strand breaks is reduced after exposure of the low dose rates, so it was open 05 Gray per minute as compared to exposure at high dose rates, 3.5 Gray per minute.

Well, this side is very interesting. The effectiveness of DNA repair system is evidenced by the lack of any reduction in the mutagenic and lethal effect as the dose rate decreases in the cell line in which the DNA repair system are impaired.

In this publication, they use a special hamster ovary cell line. This cell line, there is an absence of repair, NHEJ. And if there is an absence of repair, you have an absence of a dose rate effect

on the induction of double-strand break.

So this lack, this lack of repair is also observed when just mammalian cells are exposed to gamma rays at zero Celsius a temperature that inhibits the repair enzymes. So the number of DNA double-stand breaks is then identical at high and low dose rates whereas at room temperature it is much smaller at lower dose rates. So dose rates determines the average time interval between physical rates it has the major effect on the cellular response. The biological effects on irradiation, mutagenesis, chromosome aberrations and so on decrease as dose rate decreases. So the biological effects of the irradiation depends on two distinct factors. First, the greater efficacy of the DNA repair at low dose rates and the probability of damaged cells to be eliminated by death.

Now about pathway signal, taking the activation, phosphorylation by ATM of the histone H2AX as indicator for radiation-induced DSBs. Collins in 2004 published, have shown that at a very low dose rates, 94 milliGray per hour, DSBs are recognized by detector proteins but not repaired because of an absence of activation of ATM. So in that sense of DNA damage signaling. Signaling of DNA damage so DMA

break depends on those rates.

At higher dose rates DNA damage signaling is taking place. There appears to be a threshold for ATM dependent signaling and DNA repair.

So DNA damage double-strand breaks signaling via ATM and HWAX phosphorylation was found to be absent at a very low dose rate, 1.5 milliGray per minute. And associated with lethality, but present at slightly higher dose rate, 4.16 milliGray per minute and at high dose rates, 750 milliGray per minute.

Dose rate changes affect genes of radiation-includes apoptosis, but not genes of cell proliferation. Thus, exposure at very low doses levels of chronic radiation may cause more cell killing than that estimated for extrapolation at higher doses and that's important to note.

Well, just to show to you several well-defined pathways exist for the repair of radiation-induced lesions, some of them with high fidelity repair, you have some examples here and some of them with low fidelity repair like non homologous enjoining. And the system depends on the dose received.

Well, I would like to present you the low

dose hypersensitivity. The first time it was published by Joiner and Joiner, as observed, in many cell types, the high lethality at a few hundred milliGray followed by radioresistance at doses over 0.5 Gray.

It involves a special enzyme, the PARP 1, poly ADP-phosphoribosyl transferase activity. So for a special enzyme, PARP 1. In effective cell cycle arrest in GS-phase cells and DNA repair.

So there is a possible role of hyper-radiosensitivity responses in radiocarcinogenesis from 0 to 100 milliGray and this possible role is not yet understood.

So it is well understood for some cell types, mortality is very high per dose unit at the onset of irradiation, during the first 200 milliGray and then falls to a very low level before increasing again.

This low dose hypersensitivity is observed in many cell types leading to a high mortality rate, per dose unit, for doses of less than a few hundred milliGray of low LET irradiation.

So the cellular defense mechanism against lethality which initially showed little efficacy become more effective during irradiation. This

initial hypersensitivity eliminates damaged cells with the mutagenic potential after low doses of irradiation. So it could be good for us to have a hypersensitivity because if can't eliminate at low dose all the cells, there is no consequence for the tissue.

Well, variation in DNA repair efficiency depend on the genetic background. You have an individual hypersensitivity due to mutations or polymorphisms of DNA repair genes in the general population, due to OGG1, XRCC1 gene.

And if you have a default in damage signalling and repair, these defaults are often associated with cancer predisposition. If you have some problem with your ATM, you have a cancer predisposition to lymphoma, to breast cancer. If you have some default with your BRCA1 or BRCA2 gene, you have a cancer predisposition to breast and ovarian cancer. If you have some trouble with Lig/V, you have some predisposition to immune deficiency.

Moreover, this variation in DNA repair efficiency depends on the differentiating status of cells and tissues and depends on age. So the pathway of signalization of DNA damage is very important for the DNA repair.

Individual sensitivity is rare and usually not detectable in population studies, so in epidemiological studies. Among patients undergoing radiodiagnostic tomographic examinations or radiotherapeutic treatments some patients have been recognized with decreased double-strand break repair capacity.

Several other studies point to the involvement of repair gene polymorphisms such as XRCC3, XRCC1 and XPD in the accumulation of genetic effects in individuals chronically exposed to exposed ionizing radiation.

But XRCC1 and lutathion-S-transferase polymorphism were found associated with radiotherapy-related malignancies in survivors of Hodgkin disease. So in case of high dose received, not low dose received.

DNA damage signaling is necessary for DNA repair. Deficiencies in DNA repair are associated with cancer. Deficiencies in DNA repair are associated with individual hypersensitivity. Deficiencies in DNA repair may cause premature aging, neurodegeneration and immunodeficiency.

Well, another slide very important. Studies carried ut with the DNA micro-array technique,

this is a French publication done by Mercier, published in 2004, in yeast shows that continuous irradiation, at a dose rate of 20 milliGray per hour, so lower than the level of irradiation that causes a detectable or lethal or mutational biological effect is enough to change intracellular signaling without modifying the genome; to active or inhibit numerous genes involved in the general metabolism and in defenses against ionizing radiation.

Such mechanism brings into play defenses at doses of the same order as those due to natural irradiation which makes it possible to reduce or prevent its potentially harmful effects.

So induction of genes is dose and dose rate dependent. At very low doses, 1 milliGray, some genes involved in DNA repair are not yet induced. However, genes of energy metabolism and oxidative stress are induced at doses 1000 times lower than those needed for the induction of mutations.

For dose, upper 20 millisievert, some other genes are regulated and genes regulated by p53 and you know that 53 is related to the cell cycle. And some genes related to p53 are induced linearly with the radiation doses between 20 and 500 milliGray and some other genes involving DNA repair are

sensitive to dose rates and others are insensitive.

So this is another publication, interesting. This is a French publication also. It's a French publication on low doses of gamma irradiation, 10 milliGray, with elicit different gene sets than high doses, 2 Gray, in normal human skin cells. So specific molecular responses are triggered in cultured primary keratinocytes from adult skin at low doses, 10 milliGray, or at high doses, 2 Gray, of gamma rays.

Using DNA microarrays, 10,500 gene probes, it is shown that among 853 modulated probes, the expression of 214 are specifically modulated by low dose, so by 10 milliGray, and 370 genes are specifically modulated by high dose, 2 Gray exposure.

Low dose specific genes, about 140 known genes, include mostly genes of homeostasis, cell communication, signaling, membrane, cytoskeleton, RNA and protein synthesis, chromatin, energy metabolism, stress, cell death and transport but rarely DNA repair genes.

Conclusion, the radiation response at low dose is rather specific and quite different from that obtained at high dose.

So another conclusion that you can have,

you cannot extrapolate from high dose to low dose if you take into account those results.

In the same publication, they found that most of low dose response genes are modulated at late incubation time, 48 and 72 hours, whereas most of high dose responsive genes are already modulated at relatively early incubation times. So the type of genes induced at the kinetics of induction at low dose of ionizing radiation clearly differ from those induced at the high dose of ionizing radiation.

Another publication says that high dose radiation of 4 Gray, you have an increase of phosphorylation of proteins involved in the cell, signalling pathways and apoptosis and that low dose radiation, 2 milliGray, you have an increased phosphorylation of proteins involved in more general biological processes as was suggested and not specific genotoxicity-related responses.

Just to summarize this part, DNA damage or modifications of the chromatin are detected by signaling proteins. The activity of these proteins is modulated by the number of lesions and therefore by the dose, the dose rate and by messages from neighboring cells.

These proteins activate phosphokinase

transmitters, in particular the protein encoded by the ATM gene and the ATR gene.

In turn, these transmitters modulate the action of proteins involved either in cell cycle control, so the interruption of which promotes repair, and DNA repair, or in triggering apoptosis.

To summarize, the dose rate as a major effect on the cellular response, in general, the biological effects of irradiation, mutagenesis, chromosome aberration, decreased as the dose decreases. This may be due to the fact that while the dose rate is low, the number of DNA lesions simultaneously present in the cell is limited. Conversely, the high dose rate leads to the simultaneously presence of a large number of lesions which interferes with the coordinated action of repair system and also increases the probability of even prone enjoining, due to the presence of several double-strand breaks in a restricted volume.

Well, just to illustrate my purpose, you have activation of several pathways. First you have an activation of MAP kinases. After activation of transcription factors like an NFkB. You have induction of cellular different genes like SOD, peroxidase and so on. You have activation of kinase

ATM, ATR which have a role on the p53 with activation of these effector proteins which have a role on the cell cycle arrest with a protein of cell cycle control on the DNA repair. On the DNA repair, proteins like BR, CR1 or DNAPK or on apoptosis, we saw a role on the proteins controlling apoptosis.

So exactly the same diagram with al the genes involved. It's a different step. First step is DNA damage. Second step, detector proteins. Third step, transmitter proteins. And then effector proteins and finally, biologic effects. And you can see that the key gene and so is the key protein is the ATM/ATR protein which are involved in the DNA repair.

Well, today with immunofluorescence techniques, here with gamma-H2AX, it allows to show induction and repair of double-strand breaks. It allows to study the biokinetic of the DNA repair. And you can see that the double-strand break can be detected in human fibroblasts at one milliGray and the induction of double-strand break in DNA increased linearly with dose of ionizing radiation. but the repair system is not linear.

When the large number of cells in the same tissue are killed or damaged, repair and proliferation mechanisms are triggered which are intended to protect

the integrity and functions of the tissue by means of intercellular communication systems, direction of a cell to irradiation, therefore seems to be influenced by the number of cells affected.

Some DNA repair systems are activated by low doses of ionizing radiation. DNA repair systems differ in terms of velocity and efficacy. In particular, the repair kinetic of double-strand breaks and the probability of repair vary with dose and dose rates. In this publication by Rothkamm in PNIS in 2003, Rothkamm didn't observe a reparation and an exposure at 1.2 milliGray. So the DNA repair system are associated with apoptosis that also varies with dose and dose rate. Thus, the number of lesions, in particular that of double-strand breaks is proportional to dose even at very low doses, at doses at a few dozen milliGray, no damaged cells are found during the following days.

So conclusion, the disappearance of damaged cells seems to result from the lack of activation of repair systems which leads to an absence of repair and to cell death, all from high fidelity repair by constitutive system. When only a few cells are damaged, this elimination strategy seems to be optimal because repair systems sometimes are ever

prone and can potentially lead to the emergency of pre-cancer routes and subsequently, cancer cells. So it's better to eliminate than to keep those cells without damage.

Hence, the cell reacts to irradiation by a global and integrated response that involves several enzyme systems which govern the efficacy of DNA repair and the probability of cell death or eliminating damaged cells. DNA-induced damage is constant per unit dose. The probability of mutation is modulated within a framework on what could be called a strategy of the least cost.

At very low dose, 1 milliGray, cells are going to die because no DNA signaling and there is no initiation of DNA repair of double-strand breaks or other complex lesions. At slightly higher doses, from 5 to 10 milliGray, DNA repair is initiated. At medium doses, 200 milliGray, DNA repair starts to be counteracted by apoptosis and DNA repair can be ever prone and mutagenic which may enhance the risk of cancer. So again with this, extrapolation from high dose effects to low dose effects do not respond to the actual reaction of living cells to ionizing radiation.

So at very low irradiation doses, if a few ionizing radiation damaged cells do not survive and

are eliminated, tissue functions are not compromised.

At higher doses, a substantia fraction of cells is damaged. Tissue functions cannot be anymore assured except if most cellular damage is repaired. And cells are allowed to survive, even if mutated and fulfil some of their tissue function. This, however, may also allow genomic instability, malignant transformation and cancer to occur. So this is the difference between low ionizing radiation doses and higher doses response.

Dose-effect relationship in radiation biology are affected by nontargeted and delayed effects. Adaptive responses, bystander effects, just an example. Microdosimetric calculations based on target size of single cells do not correspond to the reality of radiation-induced effects.

Genomic instability. Low dose hypersensitivity, we saw that before. Hyperfast early cell responses and so on.

First adaptive radiation response. The existence of an adaptive response is no well established. The first low dose of radiations leads to a reduction in the mortality of organisms in vivo. But also, the number of mutations and the rate of neuroplastic transformation caused by a second

irradiation carried out during subsequent hours or days.

Priming doses of less than 5 milliGray or greater than 200 milliGray yield very little adaptation. This inducible and transprotective effect seems to occur also in humans. There is a different example, adaptive response on the micronuclei production in human fibroblasts after a priming dose of 1 milliGray and a 2 Gray challenging dose has been observed, but needs to be confirmed.

Induction of adaptive responses in human lymphocytes appears to be quite variable in different individuals. There is a publication of occupational exposure of 2.5 milliGray per year for up to 21 years resulted in variable adaptive responses in lymphocytes challenged with 2 Gray.

And one hypothesis is that genotoxic physical agents, so solar, UV and ionizing radiation, were present when life appeared on earth and very likely at that time irradiation as generally more intense than today. Recent work, as revealed, seek efficacy and multiplicity of different mechanisms which developed during evolution. Many of the systems are targeted against reactive oxygen species produced by radiation.

So the molecular mechanisms of adaptive responses are not yet well understood, especially for both priming and challenging doses of 1 to 50 milliGray.

Second nontargeted effect is the bystander effect. In multi-cellular organisms, in particular vertebrates, the fate of an irradiated cell depends upon signals emitted by neighboring cells, gap junction, bystander effect, contact inhibition, proliferation control mechanisms by means of cytokines.

Normal cells appear to be capable of inhibiting the development of potentially malignant clones. Conversely, nonirradiated cells can become cancerous in the vicinity of highly irradiated cells.

Besides an inhibitory effect, such as contact inhibition, or a stimulation of cell division, intercellular relationships can also elicit damage in neighboring cells, which have not be irradiated. This is known as the bystander effect.

The influence of intercellular interaction on low dose repair radiosensitivity suggests that there is a link between this phenomenon and the bystander effect.

The bystander effects originates from

potentially genotoxic signals sent to neighboring cells. From some of them often cell to cell contacts are required, but some cell bystander effect are obtained without cellular contacts.

The bystander effect may be beneficial or detrimental depending on the cell type and the range of doses analyzed. J.B. Little in 2000 showed for very low doses of alpha particles that more mutation of the spontaneous type were induced in the very low dose range, whereas there were only very few deletions induced. Conversely, another example, after exposure to low-dose x-ray, it leads to the death of cells in which the repair of DNA damage is defective.

So it is possible that bystander effects lay a role below 1 to 5 milliGray where few cells are actually damaged by irradiation. Are there bystander effects in vivo and in radiation therapy? What about abscopal radiation effects? Yes, they may arise, but they need to be clearly defined before assuming that bystander effects affect radiation-induced carcinogenesis.

So this bystander signal has many consequences for the un-irradiated cells, apoptosis, induction of genetic instability, delayed cell death, mutations that are in 90 percent of case points

mutation would suggest that they're induced by reactive oxygen species. So you can imagine that the reaction after exposure to ionizing radiation is not only the reaction of the cell, but the reaction of the tissue and that's very important to note.

Carmel Mosocil suggested that the bystander effect could induce in the neighboring cells an adaptive response similar to that induced by prior radiation. This effect on the neighboring nonirradiated cell could therefore, depending on the context have either productive or harmful effects. They are not proportional to the dose, but on the contrary, appear to diminish with increasing doses.

Another nontargeted effect is radiation-induced genomic instability. The definition is ionizing radiation generally changes that become apparent in the descendants.

Genetic instability is influenced by the p53 ene. It can be reduced by free radical scavengers. It is apparent at low doses and occurs at a frequency of about 3-9/1000 cells per cell/milliGray after x-ray involving.

We observe point mutations, chromosomal aberrations, telomere loss, giving rise to nonreciprocal translocations. And it has been

observed that it is associated with ionizing radiation-induced leukemia, depending on the mouse strain and to DNA repair defects with DNA-PKCs.

So an excess of leukemia in A-bomb survivors appears to correlate with excess of complex chromosome aberrations, translocations, and possibly associated with telomere dysfunction, particularly in patients with Hodgkin's disease. And this process seems to be saturated at 10 to 30 percent at low doses.

So the influence of genomic instability on the low dose-response relationship for carcinogenesis is not yet well defined.

Belakov has published non-targeted effects of ionizing radiation may have also positive consequences. Non-targeted effects of ionizing radiation might be interrelated and possibly have a protective role under in vivo conditions. These effects might relate to adaptive response because of increased non-targeted differentiation in irradiated samples.

Based on these experimental data the authors proposed a theory that the main function of the non-targeted effects is to decrease the risk of carcinogenesis in a multicellular organism exposed to

oxidative damage including radiation induced.

Well, dose-response relationships for radiation induced mutagenesis are not precise at very low doses below 20 milliGray. Gene mutations are induced linearly or with a linear quadratic relationship down to 200 milliGray. Linear non-threshold responses were observed in mice, except reverse mutations down to 10 milliGray. Induction of chromosome aberrations, dicentrics in human, is linear down to a maximum of 20 milliGray and for translocation down to a maximum of 50 milliGray. This adds to the difficulty of extrapolating genotoxic radiation effects down to very low doses.

But in fact, the lack of validity of the LNT relationship for chromosome aberration at low doses with low rates of radiation is not surprising. Why? The occurrence of a chromosome aberration is much increased when there are two or more DNA double-strand breaks in the same chromosome or neighboring chromosomes, making it possible that the rejoining of the fragments either does not restore the molecule to its initial condition.

So you know that when you are exposed to a degradation, this is a round of irradiation on the DNA. So the probability of such error-prone enjoining

therefore depends on the number of breaks simultaneously present in a limited volume and therefore decreases markedly with dose rates and is not proportional to dose, but to the square of the does. So LNT cannot be used to predict chromosome aberrations for very low doses. And a threshold is conceivable at this level.

So below 10 milliGray the biological responses are less clear. In this very low dose range, there is a much more sensitive interplay of biological processes and phenomena than at medium, so 200 milliGray, and high doses of less than 1Gray. In other words, at very low doses, below 10 milliGray, many different biological processes are activated or modulated, whereas at higher doses main stream processes like cell cycle arrest, DNA repair or apoptosis become predominant and fully determine the cellular radiation responses.

So we can try to have an abstract at this part. At high doses gene induced concern maintenance of genomic integrity. Cellular programs are directed to get cells survive, even at the dispense of error-prone repair, or to die with apoptosis or mitotic death if the mutation is an incompatibility between the mutation of cell and the cell cycle.

So responses are directed by relatively few parameters such as number of cells hit in the issue, activation of genes involved in DNA damage, signaling and repair and/or initiation of cell death pathways due to excess of damage.

At low doses, genes inducted concern general metabolism and broad spectrum responses. Many factors and parameters can interfere with the regulatory network of the overall response. The responses are very sensitively linked to cellular reactivity: sensoring and detection of changes in structure and function of important cellular constituents; metabolic states, redox and energetic states; state of differentiation; cell cycle progression, cellular communication.

For risk evaluations, the qualitative and quantitative influences of these cellular factors and parameters have to be defined. Genetic and physiological predisposition of cells and tissues, state of differentiating, and so on.

A new concept in radiation biology emerged. Cells respond even very low radiation impacts. The response to ionizing radiation involves activation of defense mechanisms, maintenance and death pathways. Cells react differentially at high

and low doses or at dose rates of ionizing radiation.

The ionizing radiation response involves activation of signaling pathways and different gene families are activated. At low doses and dose rates a multitude of parameters influence the cellular fate, whereas at high doses and doses rates cellular responses are more directly channeled towards survival, genomic instability and malignant transformation or cell death.

So the conclusion of this part, recent data demonstrate that mammalian cells react differently at different levels of dose and dose-rates of low LET radiation with DNA damage signaling, gene induction, DNA repair and apoptosis.

Second. These differences in reactivity are consistent with practical thresholds observed at very low radiation doses, below 20 milliGray, but are inconsistent with the LNT hypothesis. At low exposure levels cells appear to have more possibilities to cope with exogenous insults, and ionizing radiation responses involved a wide ranging metabolic network. Cells are generally better protected at very low than at high dose levels. And thus, human risks are likely to be lower than expected from LNT calculations.

Third. Adaptive responses. Radiation

hypersensitivity by standard effects and genetic instability preferentially expressed at very low doses, are likely to influence dose-effect relationships for mutation induction and carcinogenesis of ionizing radiation at low doses and dose-rates, but the mechanisms involved and their actual quantitative impact need to be clarified.

And last, mutation and polymorphism in DNA damage signaling and repair genes are very important for individual responses, but do not allow extrapolation to general population responses.

I would like to add a few words on carcinogenesis. A few years ago when I had present to my students the carcinogenesis process, I showed the conventional model which analyzes a series of stages. Modification of the genome which confer a selective advantage on the cell during carcinogenesis. We now know that this phenomena cannot be described by a linear process which successive genome damages accumulate at random.

Carcinogenicity is a phenomenon that cannot be reduced to a series of mutations due to indefinite stochastic lesions occurring in the stem cell. Indeed, it affects all aspects of genome function. The association of genetic and epigenetic

mechanism is just an example -- just as an example, that we know well.

So this association of genetic and epigenetic mechanism is now well established. The process leading to the transformation of the normal cell into a tumorous cells is interpreted as a Darwinian selection process, determined by a series of genetic, epigenetic events, each of which gives the cell a selective advantage in terms of survival or proliferation within the tissue to which it belongs. So it's a global response. The cell, the tissue and the body all have defenses against carcinogenetic processes and they must be successively overcome for carcinogenesis to occur.

This interaction, on-going and plays a crucial role in tissue construction during the renewal of certain tissue and the repair of damaged tissue. You need to keep in mind that contribution of multiple interaction between the cell hosting a potentially oncogenic event and its neighboring cells of the same type, the extracellular metrics are important. The significance of epigenetic mechanism is well no documented.

So if the cell, tissue and body al have defenses against carcinogenic processes and this must

be successfully overcome for carcinogenesis to occur, there are intracellular systems of proliferation control by suppressor genes and mechanisms involving the death of cells that tend to eliminate or prevent the proliferation of cells.

At the whole body level, escape from the immune surveillance responsible fr eliminating tumorous cells is based on the selection of cells that are capable of escaping from it. And you know some examples.

A good example is turmeric cancer. You know that today, we observe a large increase of tumor than before, but you know that just only a few of them will continue to increase and we have a lot of very small tumors and will stay like this without problem. It's exactly the same example with the prostate cancer. You know that we have a large increase of prostate cancer in the population and with the aging process, we have a large increase of prostate cancer, but for some of them, some men who have prostate cancer, but without trouble, will stay in the prostate without trouble because there is an immune surveillance. And for some of them because there is an escape for the immune cells we will have a proliferation of the cell.

So my intention was to show to you that if you take into account only the cell response, it's not enough. We have to have a global view on the carcinogenic process.

So initially, it as thought that the radiocarcinogenenic process was initiated by specific genome lesions and could be considered as a stochastic risk due to a rare event caused by the random occurrence of the legion inside the target.

Today, this model was gradually substituted by that of an include complex reaction dominated by intra- and intercellular signaling mechanism and largely dependent on oxidative mechanisms. They are sensitive to the micron development and to the interaction between initiated and healthy cells.

With regard to the dose effect relationship, the main contribution to progress has come from biological research. The new data reveal the complexity and efficacy of defense mechanisms against genotoxic physical and chemical agents, at the level of the cell, DNA repair and apoptosis of the tissue, role of neighboring cell and of the wall body with the immuno-surveillance.

If we have a look on the different steps

of the cell, the tissue and body defenses against cancerization, first you have intra-cellular system of cell proliferation control. You have death of initiated cell which have escaped to a safeguard mechanism like a apoptotic response.

You have control for neighbored cell, secretion by neighbored cell and stroma of regulation factors, inhibitor of proliferation.

You have bi-standard effect, exchange of signalization and regulation molecules by intercellular gap junction.

Finally, you have mechanism of immuno surveillance. Healthy cells inhibits the development of potentially malignant clones.

The cell response therefore seems to depend on the dose, about ionizing radiation on the dose, the dose rates, the cell type and on the concentration of damaged cells.

So if I would like to summarize our approach this morning and we can divide in three different area. At low dose, this is the area of the elimination. We tried to eliminate all the cells which have some DNA damage. Is that true for low doses? After we have the beginning of the reparation and the more the dose is increased and more the

reparation is important. And of course, if at the low dose it's easier to repair, most of the dose is important, it will be difficult to repair.

At high doses, the proliferation is important because you lost too much cell and as far as the tissue, it's important to have a proliferation of cells and you know that if you need to proliferate yourself, you have a higher risk to develop a cancer, so that's why we think that it's not possible to extrapolate from high doses to low doses.

You know that there is a new ICRP draft. This slide is not my slide. It's from ICRP, from people from a committee, from a Japanese man from committee to advise ICRP and I was very surprised to read this, so I give you the same side. He wrote that ICRP is very careful in using LNT, collective dose and cumulative dose. And you will see in the last draft of ICRP that NT is to manage risk from radiation exposure. And personally I have no trouble with that. We use this and that's true. And it's easy to manage the risk in a nuclear power plant with LNT. But not to assess the risk is different.

So LNT is good for managing, not for assessing the risk. And in the same draft you will see that in the case of low individual doses with wide

geographical areas, long time scales, the use of collective dose for risk estimation is not reasonable and should be avoided. That's all.

He wrote -- it's not my slide. From ICRP point of view, ICRP it's a pragmatic, realistic and conservative approach and they use NT as a tool, not truth, supplemented with real data. And BEIR VII, much more theoretical, idealistic and radical LNT as science based mainly on theory. That's why this Japanese guy takes a sentence from the BEIR VII on page 30, "The Committee concludes that the current scientific evidence is consistent with the hypothesis that there is a linear, no-threshold dose-response relationship."

So I would like to give you a few conclusions. While LNT may be useful for the administrative organization of radiation protection, its use for assessing carcinogenic risks induced by low doses, such as those delivered by diagnostic radiology or he nuclear industry, is not based on valid scientific data.

All the data show the lower effectiveness of low doses and dose rates. Moreover, the quantitative discrepancy between the results of the various epidemiological and animal experimental

studies supports the view that there are several dose-effect relationships rather than only one.

Their parameters depend on the type of cancer, the type of ionizing particle, radiation dose, dose rate, fractionation of irradiation, species, breeding line within the same species, target tissue, volume irradiated, age, and individual sensitivity factors.

Epidemiological and biological data are compatible with the existence of a threshold, but cannot today demonstrate its existence or assess its value, somewhere between 10 and 60 millisieverts.

The concept f collective dose cannot be used for evaluating the cancer risk in a population and that's very important to note.

So if I can in order to prevent radiation exposure from becoming unmanageable due to lack of knowledge, I think that research and knowledge must come up with the most effective solution to deal with risk.

So thank you for your attention and you will find the French report on the Net with ww.academie-medicine.fr and www.academie-sciences.fr.

Thank you.

CHAIRMAN RYAN: Dr. Le Guen, thank you

very much for a very information and thorough presentation of al the issues.

I may suggest that we take a short let's say 10-minute break just to give everybody a chance to stretch. We've been going for a good almost two hours now and then we'll come back and have questions from the Committee and discussion your presentation with you and we'll proceed from there. Is everybody okay with that?

So we'll take 10 minutes. Please come back right at 20 minutes after 10 o'clock. Thank you.

(Off the record.)

CHAIRMAN RYAN: I would like to start with questions from the Committee. And I will start to my left. Professor Clarke?

MEMBER CLARKE: Mike, I do have a couple of questions that relate to how this parallels some of the things that are being done on the chemical carcinogen side. I don't want to distract us too much. Should I pass and --

CHAIRMAN RYAN: No. Please go ahead.

MEMBER CLARKE: Okay. Michelle, could you put up -- I think it was slide 72. Oh, I'm sorry. Your dose response curve.

DR. LE GUEN: This one?

MEMBER CLARKE: Yes, that be fine. And I would like to frame these questions from the standpoint of a former practitioner who followed procedures for chemical risk assessment to develop information for cleaning up contaminated sites. So it's a little far afield from this.

DR. LE GUEN: Yes. My intention was not to have a risk management on this, just to give what was the most important pathway, what was the most important reaction of the cell that we know today because, see, it's a competition between all the -- after exposure. And we think that the most important pathway at low dose is elimination of cell and repairing after 20 milligray and after -- so my intention with this slide was just to summarize all the apparatuses that I try to --

MEMBER CLARKE: I understand, sir. I just want to use it to frame the question. Let's suppose that what we have to do for purposes of doing the risk assessment -- and we're going to assume a linear, you know, a threshold model.

And, again, I'm asking from the chemical side, not the radiation side based on my understanding of how that is done. So what we are looking for to do this is we are looking for the slope of that line at

very low doses.

And so this chart -- let's say we've got -- and let's say it's a result from animal testing data, again, at a very high level, not at the molecular level at all.

So on the *y*-axis, we have frequency of response, say, for tumors and laboratory animals. And on the bottom, let's say we have dose of benzoate pyrene, which is a known human carcinogen, and our data are coming from high doses, well, say to animals. And so they're up there with the red dots. And we want to somehow extrapolate that data down to zero, linear, near zero, so that we can use the slope of that line to do our risk assessment.

Now, on the chemical side, when you have something like DDT or benzoate pyrene, what we found is that the high dose data really doesn't matter which model you use. As you know, there are a number of models. And they all tend to pretty much behave the same way up at the high dose. Is that your experience at all with --

DR. LE GUEN: That's true. That's true.

MEMBER CLARKE: Yes. But as you take them down to lower and lower doses, they diverge. They diverge by orders of magnitude, which you showed on

your slide where you had the linear and you had the quadratic. And they are, as you know, multi-stage in many other models for chemical exposure.

Our challenge is to pick the right model. Now, on the chemical side, it moves quickly under the regulatory arena because our Environmental Protection Agency picks that model and tells us what slope factor to use.

And I guess my questions are, if we didn't have that constraint and we were looking at the models and we were trying to pick the best one to get this slope down at very low doses, the information you are generating at the molecular level is really what we need, is it not, to differentiate among those models or how would you do that?

DR. LE GUEN: What do we need? What is the --

MEMBER CLARKE: Well, it is, how would you advise us to pick those, pick which model is the best model to use down at very low doses.

DR. LE GUEN: My feeling is because we live on Earth and because we have a long experience, because we are exposed to a lot of genotoxic stress, I use the rendition it is not the only stress to the cell. And due to the evolution of man, if today we

are here in this room, it is because we have a long adaptation of the cell, and so a long adaptation of the defense, and that, in fact, for the moment I think it's difficult to propose only one mother.

And my feeling is that we know that the mother will be different from one exposure to another and that my intention this morning was to demonstrate that it's a mistake to extrapolate from high dose to low dose because I show to you that the reaction of the cell is completely different.

So that's true also that for in some publication, in particular with NML data, they formed a non-basis. But one of the problems today is not to say if there is or not a non-basis. It is to try to assess the risk and try to say when the risk becomes negotiable because it's not because you can avoid a few milligray so that you are not exposed to natural radiation. That's a natural background. And that wasn't the problem today.

That's why I give to you the example of radon. Of course, we know that with radon, you have an increase of cancer at high exposure, but the problem is when we need to stop to manage those risks. And that's a problem.

And we don't believe that there is a

linear approach. And we know that, of course, there is some negligible dose. And my intention was to -- I am not a regulator. I try to give all the scientific data -- and you are the regulator -- and try to convince you to have a pragmatic approach and to say that I know that DOE has accepted to put some money on the table to say, "Well, we need to have more information on low-dose exposure." And this is also my feeling that we need to continue on this field. And it's not because we need to continue that there is a real danger, a real problem at low dose because if there was a real risk, it was not possible today to be here with you.

MEMBER CLARKE: Thank you. I think you have raised a number of points in your presentation that are very appropriate to the things that we are wrestling with on the other side as well. Thank you.

CHAIRMAN RYAN: Okay. Ruth?

MEMBER WEINER: First of all, thank you very much for a very excellent presentation. I had occasion to read both the report and the paper by Aurango and Turiana earlier. And this was a wonderful addition to it.

Looking at the slide, we are not regulators. We advise the regulators. In our

regulations, we are essentially in the United States sort of forced to set a standard, some kind of a standard, and say this standard -- and the way some laws read, it says this standard protects most, but not all, of the population.

Where would you -- I recognize this is a terrible question. Where would you set such a standard? What would be your opinion if you were in our position of advising a regulator?

DR. LE GUEN: Well --

(Laughter.)

MEMBER WEINER: Let me ask it a little better. Looking at your graph, would you set it somewhere in the region of 10 to 20 millisieverts?

DR. LE GUEN: Yes, I think, but, you know, what is the reality? What is the real exposure of the population is not 10 or 20 millisievert. It's lower than this. We are at the labor of natural background.

It's very difficult to say, "Oh, the risk is negligible" because it's impossible to say that you have a higher risk with just this few little small doses; in fact, when you know that the natural background is much more important than this.

So that is very important to keep in mind which kind of dose are we talking here. Is the dose

-- because for me now, if I try to give you some example, not in the medical field because that is a real problem, but for nuclear workers and for population who live in the vicinity of NPP. So those are so negligible that it's not a real risk.

The problem is to -- I believe because I am a physician -- I forgot perhaps to mention this -- that today that's why I wanted to show this slide. We need to check to optimize in the medical field the number of chest X-rays on all examinations that we have to do.

Particularly I would like to make some difference between adults and children because we know that the people who are sensible to radiation are the children. I would like to say, "Well, be careful if we need a force because there is a balance. If we need some medical examination, it is because there is a disease and because we can't -- there are benefits for the patient, but it's important today to avoid to multiply the medical examination and particularly one where you have small children."

For others, it's not a real problem because we know that the sensibility is not the same. And so it's much more my approach then to say there is only one curve and say it's only one approach and for

all the world population. I try to see which kind of dose we need to manage and which purpose. Is it in the medical field? Is it for the population?

And so that's why the answer is much more complex than just to say, "Well, take this. And that's all."

MEMBER WEINER: Thank you for that.

I have one more. You didn't dwell on cumulative effects.

DR. LE GUEN: Yes.

MEMBER WEINER: And I wonder if you could say something about if what has been observed in cumulative effects of low dose. If you get a low dose today and another a year from now, do they add?

DR. LE GUEN: That is a key point, of course. This is a problem of the sensitivity and the consequences of a chronic low-dose exposure. We know, of course, that the accumulation of dose is completely different from an acute for the same level for an acute dose received. And because we have some mechanism, we tried to show to you that we have a very low dose or we have the opportunity to repair the damage or we have the opportunity to eliminate all the cell exposed to ionizing radiation.

So about a chronic exposure, I show to you

also the problem of sometimes you have this kind of question of hypersensitivity and what kind is possible to propose to this population, which are sensitive to ionizing radiation.

In fact, this is very important to which kind of people, which people are we talking. Is this people we have polymorphous sensitivity? And we know that in this case, if there is sensitivity, it's not at low dose but at high dose.

So it would be interesting if there is a cancer and you want to treat the cancer if you know that those people are sensitive to radiation to have a practical approach and if you have the possibility to have a choice between a chemical approach or radiation approach to take this because this is a sensitivity at high dose.

Today we have no problem because I showed to you that we have very low dose. For the moment, there is no data, no epidemiological data, to prove that there is a consequence of hypersensitivity for a subgroup of people. And, in fact, it's today, for example, for nuclear workers and so on.

We have no rule to say to say, "Well, you are sensitive. You can't work because you are sensitive to radiation." It would be not good to say

that because first we don't know. And, moreover, you say you cannot have a job. And it is not true. So it's important to make a difference between the person's sensitivity and the real dose received.

Remember my slide today on nuclear workers. Dose received is about 1.6 millisieverts on average, so a very low dose. And so that is very close to the natural background. It's impossible to say that this dose of 1.6 millisieverts you will have higher risk than with the same dose due to natural background.

MEMBER WEINER: Thank you.

CHAIRMAN RYAN: Professor Hinze? Bill?

MEMBER HINZE: Well, I want to thank you for your presentation. I am trying to put some of this into my own framework of knowledge. The cell response is similar to what we might call a seismic response. And one of the things that is very important to us in seismic response is the duration of the seismic vibrations.

And when I look at your list of the factors that are controlling the cell type, the dose, the dose rate the cell type, and the concentration, as you have listed there, duration, is that part and parcel of this?

DR. LE GUEN: Duration of what?

MEMBER HINZE: Duration of the dose, the duration of --

DR. LE GUEN: So dose rates?

MEMBER HINZE: In other words, is this part of the aging process?

CHAIRMAN RYAN: The exposure time.

MEMBER HINZE: Exposure time duration. Is that part of the cell --

DR. LE GUEN: We think in some publications, yes, of course, it is one parameter.

MEMBER HINZE: And so it is part of the cell response, --

DR. LE GUEN: Oh, yes, sure.

MEMBER HINZE: -- the length of time, the duration?

DR. LE GUEN: You're right. There is a slide. And this is a French publication when we have served -- oh, that is a good question because when you make some science, you say, "Well, I have some cell. I would like to have a kinetic of the answer, of the cell." And you say, "Well, I would like to see the answer after ten minutes after exposure." And you observe something.

But if the kinetic is completely different

and is related to the dose received, you can say, "Well, there is no trouble. There is no response." But it's no because you have no response because the response was later. You have not an earlier response.

Is that exactly perhaps what you mean?

MEMBER HINZE: Part of it, right.

DR. LE GUEN: Yes. And the response of the cell can be completely different from the type of cell. Of course. But so of the dose received, that's true today. We know that in the case of low-dose exposure, the response was not -- it would be not an earlier response but a later response, after one or two days and because it's not the same gene and so on. So that's true.

MEMBER HINZE: Let me ask you a question that perhaps isn't fair, but LNT has been with us for a long time.

DR. LE GUEN: Yes.

MEMBER HINZE: What do you think is the strongest evidence for LNT? And why do people still use the linear no threshold in the face of the accumulating evidence from biological research?

DR. LE GUEN: Well, you know, personally I have no problem with LNT because when I say in my proposal if we need to manage people, it's an easy

line. We can do it. The problem is all the -- perhaps did you see the publication about Chernobyl and the consequences in Europe after Chernobyl?

MEMBER HINZE: Yes.

DR. LE GUEN: When you use the LNT approach and you say, "We can calculate number of deaths in the next future because we take the cases" and you know that perhaps is not true and to say you can say to the population, "Look, due to this dose, we will have an increase of the cancer."

And I say, "Well, okay. We can. I have no problem." And in France, we have RTDF, for example. We have no problem to use LNT, but we have a problem if we use this hypothesis and to say this is true and we can access the risk with it. And that's not true. Is that not fair and that not true?

MEMBER HINZE: What I have learned from your presentation and your publications is that we must be very concerned about using population statistics. And this is for a variety of reasons.

It is much better for us to use the results of the biological studies of the cell DNA and so forth. However, I think there is a certain comfort to the population at whole to rely on population statistics.

I'm wondering if the knowledge of the mechanisms going on in the cell and the related tissue will bring us to the point where we can design a population survey that will show the kinds of effects that you have talked about at these lower levels.

Is that possible? Is that possible now or is that something in the future?

DR. LE GUEN: Why not? Why not?

MEMBER HINZE: Would you design --

DR. LE GUEN: In France today, it --

MEMBER HINZE: How would you design a population survey?

DR. LE GUEN: For me, you know, the precedent showed this with a monitoring, a long-term monitoring, after Hiroshima-Nagasaki exposure. In one of the last publications on this cause, it demonstrates that, of course, if you take the world populations, it's not good to assess the risk because we know that if you are young when you were exposed, the risk is higher than if you are an adult.

So there is a difference between -- it's important to take not one group but a different group if you are a woman than if you are a man, for example, with breast cancer that you know that since the last ICRP publication, not the last but the last draft,

they proposed after Hiroshima and Nagasaki monitoring to increase the WTs or higher sensibility of the breast tissue because they observed that there is a -- so, of course, it's important to and sometimes not so easy to have different groups.

And what is the definition of radiation protection? To protect the most sensitive people. So if you can protect the most sensitive people, we protect everybody. And for the population, I think that's important to protect.

And, in fact, if you have a look on the regulation, when we talk about one millisievert? What is one millisievert? It's not a lot. And with one millisievert, we protect all of the population.

MEMBER HINZE: Thank you very much.

CHAIRMAN RYAN: Thank you, Bill.

Allen?

DR. LE GUEN: The question is up. And, of course, that is important to continue to work on this field and to answer all the parameters that we don't have today.

MEMBER HINZE: You know, I was trying to put your talk, your excellent talk, into my framework. And one of the things we do is we learn more about the process, --

DR. LE GUEN: Yes, yes.

MEMBER HINZE: -- the science behind them. And then we can design better experiments. And that, it seems to me, is what we can do in this field as well.

DR. LE GUEN: And to imagine that ionizing radiation is not the only stress for the cell.

MEMBER HINZE: Yes. We have to --

DR. LE GUEN: It's because we have defense mechanism against the stress for the cell. And the answer to ionizing radiation is an example of the answer for the cell, but the cell is much answer for the genotoxic due to the food, due to the chemical. We talk about the chemical product and so on. And, of course, it's because we have not different mechanisms. We have only one but directly related to the dose.

MEMBER HINZE: Thank you.

CHAIRMAN RYAN: Allen?

VICE CHAIRMAN CROFF: I am going to try to ask an intelligent question here. Thank you for a comprehensive description of the science and radiation biology here. I will admit it's not my field either.

Noting that we are advisers to regulators and the area in which we regulate is doses, you know, whether an individual gets perhaps 200 millisieverts

in a lifetime or 210 or something like this. We're dealing in the 200 to 300 range.

Given that that that's the dose, area of dose, in which we have to regulate, we're stuck with that natural background is I guess what I'm saying.

What is the implication of your science or what is the science you have described telling us about the dose-response curve in that area?

DR. LE GUEN: In fact, it is the same question that Ruth said before about the chronic exposure and at the end you have -- is this what you mean?

VICE CHAIRMAN CROFF: Well, I am assuming the exposure is chronic, that it comes in --

DR. LE GUEN: Yes. And that's life.

VICE CHAIRMAN CROFF: That's life. I want to be clear of what the science you have described is trying to tell us. Is it trying to tell us that it is linear in that regime or does it not support that?

DR. LE GUEN: It is difficult to answer. You know, you remember what I said before? It's not the cell reaction which is important. This is the outcome of the cell. And if at low dose, a chronic low dose, you can all repair or you can eliminate the cell, there will be no consequence for new exposure

because there is no cell.

The problem is when you need to accumulate mutation and the operation is -- is it possible that due to chronic exposure and all along your life and we can accumulate mutation? No, it's not this because this is exactly the aging process why we observe an increase of cancer due to the age. It's because time with a long time at the end. We know that the immune surveillance is not the same way we are ordered when we are young.

So the difficulty is to say, "Well, we know that at low dose, we think there is no real consequence because we can manage this dose" and at which level it will be difficult for the cell because we have perhaps no problem the first time, but due to a long-term exposure, we will accumulate mutation and so on.

And we think that today because for me 20 or 50 millisieverts at this level is quite the same dose, not for the regulation because we know that in Europe, we adopt 20 millisieverts. I'm talking about the consequence of the exposure.

If we respect, for example, for nuclear workers, there is no problem because we are at a very low dose. But the difficulty today is to give a

number. And I cannot give this.

I understand that it will be so easy to say, "Well, below this number is no problem." And, in fact, it's not so easy to say. So that's why I have some difficulties to answer to your question.

I know that, in fact, if we have a real dose exposure, it's because we know also what is the natural background. I give you the example of the KALA and the RIA, where the natural background is so high. And because we know that, we did not observe an excess of cancer.

We can say, "Well, if we have a look on this publication, we can say, 'Well, there is no risk.'" But it's only one exposure, one example. And so because I am a physician and because I am scientific, I say, "Well, that's a good question." But it seems to be, but I have not the proof, the real proof. And it's difficult to answer this.

But if we respect the levels, the real levels, the low levels, that we have today, it seems to be -- so there is no real risk. And we don't observe. There is no excess.

VICE CHAIRMAN CROFF: Okay. Thank you.

CHAIRMAN RYAN: I am trying to think of a few summary messages that we can take away from this

morning's discussion.

DR. LE GUEN: Yes.

CHAIRMAN RYAN: And I take Professor Hinze's comments. He has really explored some of these variables by a seismic analogy, which I think are really helpful. Thank you very much.

It strikes me, too -- and a thought entered my mind when Dr. Weiner was asking her question and Dr. Clarke as well. The one aspect of radiation protection that might be a little different is that we have this overriding principle of ALARA in the U.S. --

DR. LE GUEN: Yes, yes.

CHAIRMAN RYAN: -- and optimization in the ICRP framework.

DR. LE GUEN: That's a good approach.

CHAIRMAN RYAN: So whatever number we arrive at, we are never satisfied with the number. And we always seek through a very formal process to further reduce exposure.

I think the French experience --

DR. LE GUEN: Yes, yes.

CHAIRMAN RYAN: -- in power plants is clear. It's been coming down. The U.S. experience when we plot the same curve is exactly the same kind

of trend where annual doses are in the two rad -- sorry for my translation to our units.

DR. LE GUEN: No, no, no. That's why I wanted to compare this approach on the medical field and to say, "Well, we need to have exactly the same approach, try to minimize as we can perform it in the nuclear field." That's true.

CHAIRMAN RYAN: So, all of that being said, I think one of the important messages that we should take away is that if you use LNT for a policy-setting approach to setting a standard for workers or for any other situation, that is not unreasonable to do.

DR. LE GUEN: No.

CHAIRMAN RYAN: But for me, the important conclusion is I remember when I first took radiation biology, we talked about multi-hit, multi-target, single-hit, single-target, and very geometric kinds of views of radiation interaction with matter, almost relying just on physics and energy deposition. Volume of DNA was important, rather than the structure of DNA, and so on.

It's a much more complicated, multidimensional problem.

DR. LE GUEN: Yes.

CHAIRMAN RYAN: There's the kinetics that Professor Hinze alluded to of dose, dose rate, dose duration. There's the physics. There's linear energy transfer, high LET alpha particles, low LET, and something in between with neutrons, protons, and the rest.

Now there's this very complicated biological dimension of responses at molecular, cellular tissue, organ, and organism levels, all slightly different and complicated.

And I think when you try and integrate all of that into one view, it is challenging at this point in time. And I take this from your presentation, all the different dimensions, to say we understand the human biology of how to deal with low-dose exposure.

But, that being said, I think all of the advances that you have reported and all of the key studies you have reviewed with us today are moving us along.

So, to me, I always separate the policy aspects of using LNT from the radiation biology and ongoing knowledge improvement that is ongoing in that area.

DR. LE GUEN: Yes.

CHAIRMAN RYAN: I think that is very

important.

DR. LE GUEN: Yes, yes.

CHAIRMAN RYAN: Often I hear people quote a radiation biology paper and say, "Oh, that means our policy should be" --

DR. LE GUEN: That's a scientist free.

CHAIRMAN RYAN: So I take away that message that we must be very careful not to use policy arguments to argue science or science arguments to argue policy necessarily. Somewhere they have got to come together, but we have got to be careful to do that fairly. And I think you have given us a fair presentation of those issues.

Am I summarizing, Bill?

DR. LE GUEN: I fully agree with that. One of the problems that we have today is the perception, the feeling of the population. When you give a number, the problem is that, oh, if there is a risk, if there is a number, if there is a risk below this number, and there is the difficulty to make a difference between managed risk and assessed risk and the perception of difficulty exists to say, "If we manage" because we know we have this knowledge today and we give some -- the regulators say, "Well, one millisievert for the population" and so on because we

try to manage the risk. And that is good.

You have seen a decrease. It's because we have decreased the dose for nuclear workers and because we have adopted an ALARA approach because we have today these kinds of exposures, the level of this exposure.

I think one of the problems that we have today is to have the difficulties to explain to the population that it's not because we give a number. It's because of the numbers that you are at a real risk of concern because the regulation is to avoid, to have the upper limit, where there is a real risk. And this is important to explain to the population that's completely different. I don't know if it is somewhat --

CHAIRMAN RYAN: I think we are on the same track, you know, thinking it's really -- I like the short way you said it: to either assess the risk or manage the risk.

DR. LE GUEN: Yes.

CHAIRMAN RYAN: That is the essence of the difference, yes.

DR. LE GUEN: That's why the French report tried to give this argument to say, "Well, we can know where is the upper limit." I presented to you the

epidemiological studies.

And where we know that there is a real risk after is just, "Oh, we can deal with the risk." And we don't know. It seems that there is no real risk at very low dose. But we need to manage this.

And it's not because we manage that there is a real risk at this level. That's exactly what I wanted to show to you this morning.

CHAIRMAN RYAN: And you did that quite well and quite thoroughly, I might add. It was a wonderful session.

Are there any other staff questions briefly? We have another presentation. Start with Latif and then Bobby.

DR. HAMDAN: Yes. Latif Hamdan, ACNW staff.

The question is, if LNT is good enough for dose management and regulations and we know enough that one millisievert is protective --

DR. LE GUEN: No. It's not a question of protection. It's a question of, of course, it is enough to protect. But one or two or five millisieverts for me is exactly the same dose.

DR. HAMDAN: But the question is, if we know all of that, why is there so much buzz about the

low and very low dose radiation research and work going on? And are any of the health physicists who are doing it for the reasons that Dr. Ryan mentioned, you know, to study the mechanisms of the cell, et cetera, et cetera, not perhaps encouraging or creating a situation where they are confusing everybody?

DR. LE GUEN: Well, I think I have a few arguments on this. But I think one of the most important arguments is kinetic risk. Why would you like to ensure considerable expense in order to limit such exposure when you know that there is no risk?

And I prefer today because we have as problem, for example, I appreciate what was the role of the government about typical consumption in the United States. But because that was a real risk and it was very important to say, "Okay. John Wayne, it was a long time before. And today we know that there is a real risk of lung cancer" to put money and to say, "Well, we need to have a good politic on this field because we will have a real result.

The problem is that one moment when you have no risk, you can continue to decrease. But you do spend money for nothing. And that's why.

DR. HAMDAN: But it's not just money.

DR. LE GUEN: Oh, no, no. I say I have

two arguments. That's just one.

DR. HAMDAN: So, you see, the point I am making is yes, there is room to do research in health physics and do it on the cell and the mechanics of the background radiation, radiation on health. There is room for that to be sure. But does it belong in regulations? Does it belong in risk management? Does it belong in administering of a regulatory agency, if you like?

CHAIRMAN RYAN: If I may, let me tell you the health physicists' view. I think it is important to recognize that the fundamental studies in cellular radiobiology have much more far-reaching effects than telling something to do with radiation protection standards. We are actually learning a lot about fundamental behavior of the cells and its many parts and pieces.

It might reveal mechanisms of cellular damage that lead to better understanding of carcinogenesis and, therefore, cancer cures. That's possible. That's a big, huge goal.

So I think it's a little short-sighted to cut it off as only having to deal with radiation protection standards. Those studies are much broader than that, although they are founded in understanding

low-dose effects.

But the radiation biology goes well beyond radiation protection. I think that's a fair statement. So I wouldn't narrow it so much. So I think there is broader value there, Latif. That's my own view.

DR. HAMDAN: Thank you, Mike.

CHAIRMAN RYAN: Okay. Boby, you had a question?

DR. ABU-EID: Well, first of all, I would like to thank you for the outstanding presentation. It's one of the few presentations I've ever heard that were so detailed and based on science.

DR. LE GUEN: Thank you.

DR. ABU-EID: Also I would like to thank ACNW for hosting such an outstanding speaker from the international community to hear the other point of view.

I have two comments and two questions if you don't mind.

CHAIRMAN RYAN: Please.

DR. ABU-EID: First of all, I would like to remind you that the low dose as follows as one of their definitions, actually, which is the low dose is defined as this, then, .1 milligray per minute over

months or a lifetime. And we see here the duration period. It was not elaborated on. So I wish this to be taken into consideration.

Other comment. I wonder, actually -- you came to two different conclusions, you in your report and BEIR VII. And assuming that the same data were used and the public and the scientific community, they wonder what are the differences, what are the bases, what are the statistical variations that you made certain conclusions and BIER VII, they came to a different conclusion. And that is really the issue we are trying to find.

The second question I would like to raise -- and I would like to be brief -- is numbers. I understand you declined to say numbers. However, I would like to hear your views about certain numbers established by ICRP in terms of risk.

The ICRP in their latest recommendation, they recommended to use 10 microsievert as a boundary between significant risk and insignificant risk.

DR. LE GUEN: Yes, yes. I know.

DR. ABU-EID: Hearing your lecture, I would like to hear now at least -- I'm not looking for a number. I understand.

DR. LE GUEN: Yes, yes, yes.

DR. ABU-EID: You decline to give numbers.

DR. LE GUEN: I know that.

DR. ABU-EID: But I would like to hear your views --

DR. LE GUEN: Yes. I know.

DR. ABU-EID: -- as a person who has been involved in this area about this number.

DR. LE GUEN: We were altogether in Prague two weeks ago. And, in fact, it was one of the questions asked because of what is 10 microsieverts? Nothing. And it's nothing. And you know, of course, of the example how many Paris-New York, we would fly. You could have very easily 10 microsieverts.

So, in fact, I don't agree with this approach and because it confused also the experts. You remember when I said before about the feeling on how because we are talking about 10 microsieverts? It's because you have 12 microsieverts the risk will be higher. No. That's wrong. That's a mistake.

CHAIRMAN RYAN: And I think, Boby, if I may add to your comment, I think it's an excellent focal point, excellent focal point.

DR. LE GUEN: Oh, yes.

CHAIRMAN RYAN: And it really shows the flaw in extrapolating a risk management strategy to a

risk assessment strategy. So they took a risk management strategy and tried to extrapolate it to a risk assessment strategy. And that's where the mistakes are made.

DR. LE GUEN: Exactly.

CHAIRMAN RYAN: Fair enough.

DR. LE GUEN: Are you able to measure 10 microsieverts?

(Laughter.)

DR. LE GUEN: What was our question, please?

DR. ABU-EID: We would like to know to the first question, which is the public arrays about the conclusion that you made versus BIER VII and both of you respected organizations. So what are the differences in the data that you used such that you come to different conclusions?

DR. LE GUEN: Well, in a few minutes, it is difficult to answer, but I can say -- do you remember during my introduction, I said, "Well, be careful. It's not the reaction of the cell. It's not the answer of the cell after exposure that is involved. This is outcome of the cell."

One of the problems today is that if you have a look at different publications, they say,

"Well, we observe this. We have this data. But it wasn't the problem. We have not the opportunity to have the global answer of the body."

And one of the differences with the BIER VII is that we say, "Well, don't look only at the ionizing radiation problem, but look all about the cancer." And that's why I say, "Well, we know we have a lot of examples about the answer, the neighboring cells, the immune surveillance, and so on."

And it's not because you have only a look on some cells and you observe something that you can't extrapolate easily to the body because there are other factors. And perhaps it's one of -- it's not because we have the same publication that we have sometimes a different view because in our group, we are all physicians. And we come from different sectors. And we have an experience on carcinogenicity.

Before, when I was at the hospital, I was an oncologist in radiotherapy. And because I have also the experience to have the opportunity to take all of this experience and to say, "Well, be careful when we have some results. Okay? This is what we observe, but what will be the consequences for the body is sometimes different." And we have to take into account all of the parameters.

Thank you.

CHAIRMAN RYAN: Thank you, Boby.

We have another request for time from Dr. Theodore Rockwell to make some comments to the Committee and to present us with some information that we have in written form. So, again, Dr. Le Guen, thank you so very much for your presentation and your interesting discussion.

Dr. Rockwell, I am going to ask that you go up to the front and take that same seat and present your materials to us.

(Whereupon, the foregoing matter went off the record briefly.)

DR. ROCKWELL: I did put some material both electronically in here so that it will be available on the record and there are copies on the back table there.

The main thing that I was concerned about this morning is that, in addition to the subject that was covered, there is a great deal of information available on the hormesis, on the beneficial effects. And if you look at, for instance, NCRP-136, right up on page 6, it says, "It is important to note that most populations exposed to radiation are not harmed thereby, low-dose radiation. And, in fact, most

populations are benefitted." And that's really important to note.

And so I have put some of that literature with some links to others because that is really important. The statement was made that it would take huge populations to demonstrate epidemiologically that there is no risk at a low level. But that premise is made on the premise that low-dose radiation follows the LNT. If it doesn't, if there is a hormetic effect, then, of course, those limitations don't apply.

And the literature shows, in fact, that, as he says, in each case, whether you're talking about the observers of bomb tests or the survivors of Hiroshima or nuclear workers or high natural radiation people, any of those things show this hormetic effect. The raw data almost always says that.

And then people scramble around to try to demonstrate that, well, there are these complicating factors and, therefore, it may not be true.

I think the real problem comes up in treating nuclear radiation as something apart from everything else. And that was a point that was so well brought out this morning. The body is subject to all kinds of attacks. And radiation is one of the

least of its problems.

We have got this situation. I think Alvin Weinberg started it with this idea that nuclear technology is a Faustian bargain. We have a wonderful gift, but there is the devil to pay.

And so we get into a situation where we say, "Well, we may not be absolutely sure that there is no risk at low levels. So what is the harm in assuming that there is a risk?"

And that is exactly the way it is expressed in a number of these documents. ICRP is particularly strong on making the statement. What is the harm in being cautious? And the fact of the matter is that there is great harm in it. There is great harm in it, not only the waste of money, which, of course, reflects in other ways. But we have situations in which our nuclear power plants are being rewarded financially and in their ratings from the NRC as to how good a plant operation they are running by reducing their collective dose.

So you have a situation that there is tremendous personal pressure on individuals to reduce the collective dose at a nuclear power plant. And you say, "Isn't that grand?" no, it is not grand. It is very easy to reduce the collective dose. If nobody

goes into where the radiation is, the collective dose will be kept closed, will be kept low.

But if you want to know is the thoric acid eating through the reactor head or is some instrument acting up, you want to send people in periodically where the important safety equipment is to see that it's all right. And if you have your management pressurizing you to not do that because you will raise your collective dose and, therefore, they will go from being a number one plant in the NRC scale to being a number two or a number three, that is working against safety. It is actually harmful to do that.

And so I think that the point that was made so well this morning that radiation is only one of the things that the body is undergoing and that if we take that one variable and treat it as if it overrode all others, we do great harm in safety and we do great harm in the public's mind as well. And so I just want to emphasize that point.

I would urge any of you who want to get further into this to look at some of the reports. We're very emphatic about the new research that's going on and the new findings and the wonderful techniques that molecular biology has fought, but if you look at one of the reports that I put into the

record, there's one written by Jim Muckerheide, who is here, the founding president of Radiation Science and Health, came down to Boston for this meeting. He has a report in there that I put in the record that says there never was a time when it was not known that low-dose radiation is not harmful. And the first report that he cites is 1915. And so this is not a new idea.

When they first started, when X-ray machines were first a new toy to use in research, it was only months later that tests were being made on using this to work on low-level infection. And right in that report back 100 years ago, they knew that the radiation dose that they were giving with these crude X-ray machines was not sufficiently high to endanger the bacteria, that what must be happening is that this low-dose radiation must be stimulating the body's defenses. They knew that back 100 years ago. And I think it's important for us to recognize that.

So the last point that I want to make that is in connection with, gee, we're regulators and what we want is a number, I think that the actual threshold if we say, "This is now the threshold and up here is dangerous and below here is safe," that's not going to be the answer. That's not the important thing.

We tried this with a below-regulatory concern in this country. And it was shot down and I think for valid reasons because what this says, really, if you back off a step from it, from the science of it, it says that there is still a danger. One gamma ray can kill you. There is still a danger, but it's too expensive to protect you from it. So we're going to tell you you should not be concerned.

That is the way it reads out. And I think that is not an unnatural reaction for people to have that situation. And if we're talking about a risk that is so small as to be negligible and if it's less than other risks that we normally accept, like flying to Paris -- I don't know anyone who would not fly to Paris to avoid the radiation. And, yet, that's the point.

Dr. Wallender, who is the former head of the Swedish Radiobiology Society and a member of UNSKIR, took the example of being in a presence of a room full of risk evaluators. And the fellow says, sort of jokingly, "Is it safe for me to stand up, get out of this chair?"

And the regulators all laughed and said, "Of course."

But the nuclear regulator says, "Oh, no."

He says, "I can't assure you that that is safe, that you may have a very weak heart, and that that might be the very thing that would trigger you off. I cannot assure you that that is safe to get up out of that chair."

I think that is the position, the mindset that a lot of our people have gotten to by putting radiation on a pedestal of being a hazard that is so much worse than any other.

So I think that we have to get to the point where we say -- and I think the hormetic studies demonstrate this, take us all the way back to page 6 of NCRP-136 -- that most populations exposed to low-dose radiation are not harmed. In fact, most are benefitted.

That says to me that at the low-dose level, there is no hazard. And there is a great difference between saying there is no hazard in a practical sense, there is no hazard, versus saying, yes, there is a hazard at any level. There is no such thing as a safe level of radiation. But you shouldn't worry about it, and we're not going to regulate it. I think that is just an untenable position. I don't think it's a responsible position.

Thank you very much.

CHAIRMAN RYAN: Thank you, Dr. Rockwell.

Any other comments or folks who want to make any observations? Just tell us who you are and who you are with.

DR. KOCHER: My name is David Kocher from SENES Oak Ridge. I guess I should reveal I'm a consultant to the ACNW, but I am not standing up here in that capacity right now.

I wanted to ask you about the Oxford survey on childhood cancer. I know this was discussed in your report. And you did not talk about it this morning. It does seem to indicate that there is an observed effect that doses may be ten times lower than where you set your cutoff.

And what this might do is it doesn't necessarily negate your argument about there's a region where problems are eliminated and there is basically no risk, but it just might lower the boundary at which that elimination region takes hold.

And so my basic question is if you would discuss very briefly the view of your Committee about the Oxford survey on childhood cancer?

DR. LE GUEN: Well, in Oxford, there is, in fact, only one publication when it says that there is a risk at 10 milligray for a fetus. This is what

you mean?

And, in fact, I would say, well, from my point of view because I am a physician, of course, I would like to protect first the fetus and so the pregnant woman.

For, example, in France in the nuclear energy field, when there is a pregnant woman, she cannot work anymore. So this is I think a practical approach.

After concerning the real risk of 10 milligray, we say, "Well, in fact, there are all of the publications. And they don't have the same level of risk." But anymore if there is a risk, we need to have all the publications to demonstrate this.

I don't say that is not true. I don't say that is true. I say, well, why not? But please give me all that are given because only one publication -- and there is some controversial approach on this. I need more explanation. So it's not my point of view. It is because there is only one.

So do you understand what I mean? So from a scientific approach, I say I need other data to prove this real risk at 10 milligray. But from my position as a physician, I say, well, it is not a problem. Because I am a physician, my first step is

I want to protect the pregnant woman. I want to protect the fetus. So I must avoid the fetus from exposure, from external exposure, and so on and particularly in the case of exposure due to her job.

So there is a balance, I think, a pragmatist's approach and reality. And the reality, there is one study. We need more data. But why not?

DR. KOCHER: Thank you.

CHAIRMAN RYAN: Yes? I'm sorry? Yes, please.

DR. MUCKERHEIDE: Hi. I am Jim Muckerheide, President of Radiation Science and Health and Massachusetts state nuclear engineer. I organized the sessions starting in '94 up to 2001 at ANS with dozens of papers and about two or three dozen sessions over those six years.

I wanted to just make a couple of observations. One was that in this discussion, the premise that radiation is damaging is true if you look at it in terms of hitting cells with radiation. And a lot of the references are to cell studies.

So the cell studies tend to always show an incremental damage. They do get repair, but they're not really the repair of a whole organism. In whole organism studies, you almost always get at low doses

pretty complete repair. You're going to get very error-free repair at that level and stimulate other enzymes and mechanisms, especially immune mechanisms that I think that are understated here except in the whole body sense.

Those aspects are really critical, I think. Plenel in France for 20 years or so in his group did a lot of work where the exposure was reduced from natural background and always saw detrimental effects from reducing radiation from natural background.

In general, I think treating radiation as a damage agent that the body or the cells or even the original formation of life had to overcome is a misperception, that there is, in fact, not so much an issue of having to protect the cell from radiation but that radiation is part of what makes the cell function.

There was a statement in this meeting or in an ACNW meeting that was a joint Committee meeting in March of '96 where Charlie Wilson came in and said, "Well, I came about this hormesis idea fairly late. In 1958, I was down at Oak Ridge," he said, "at the lab. And we were doing experiments where potassium had been taken out of, potassium-40 had been taken out

of, potassium. And the potassium was used as a source for cells. The cells looked okay, but they didn't function.

And so this whole process, including other studies where potassium had been removed, Don Luckey did that at Argonne in '86. And there's a paper in *Rad. Research*. If you take the potassium out, there is a loss of function within the cells.

Without potassium-40 in the potassium, you could bring in an external source. And the cells would recover. So, you know, put a thorium source into the enclosure, where it's being shielded. And having had its potassium removed, you can add the potassium-40 part of it back. You can add the potassium, natural potassium, back into the mix or you can just give external exposure and the cells recover.

In small organisms, for example, there was a situation. There was a serendipitous experiment in the literature where two sets of organisms were growing differently in two slides that were essentially identical slides. After a lot of study and investigation, they found there was more thorium in one slide than in another slide.

So this idea that there is radiation is only in this damage mechanism and is not actually an

essential aspect of what we require for cells to function, for life to function I think is understated in a concern about how much damage radiation does.

Just a couple of other observations. I like to point out in the Russell case about the LNT and applying it to mutations. Back around '96 or so, Paul Selby at Oak Ridge, who is a geneticist, who is a member of the U.S. delegation to UNSKIR, Paul Selby, who had been doing some work for Lee Russell, found that they hadn't counted all of the control mutations. And when he brought the control mutations, -- this is in the '52 to '54 time frame -- when he includced the control mutations, the whole idea of doubling dose was changed to the point where the doubling dose would have been more than a lethal dose. The whole LNT that was kind of built on from '56 on as a function of coming from Mueller and radiation damage for genetic effects is without foundation as well as having no foundation in carcinogenesis.

Another point was that the cellular responses are really misleading in the way that the responses don't fully take account of repair, but in *ex vivo* studies, you can get some of that fixed. But in *in vivo* studies, looking at some of the same cellular kinds of responses, the stimulation of a lot

of the repair mechanisms and, in fact, the net beneficial effect is much more readily seen.

So I think both in the ICRP and the BEIR and even in this discussion, there's too much reliance on moderately straight lines in cellular experiments.

Those are just a few comments on the general conclusion. I would very much stress -- and I have done this with Turiana and Roland Mass after the paper was written and I had commented on their English version before it was released -- the whole immunological issue is not adequately addressed. And they said, "Well, next time we're going to be working that in because we haven't really had the wherewithal to incorporate it."

And I think really addressing immunology in the context of all of this *in vivo* work, including the reduction of cancers and other diseases from the early work, is really critical.

Ted referred to the 1910 work. There is a 1920 paper in PNAS by Murphy at the Rockefeller Institute in the Journal of Experimental Medicine, which is one of the papers of that series of about ten years' worth of work by Murphy and a number of others there that essentially found -- they were investigating immunology and cancer. And they were

looking at it in terms of a number of factors.

They have this one paper. It's just four pages. But they were looking at physical effects. And one was radiation. The other was heat. And they both have pretty much the same effect. And that is when they had a low dose, a moderately low dose, they began to suppress the lymphocytes. And, as they did, whether they were injecting cancers or self, you know, putting cancers back into the animal, they were getting increases in cancer.

When they brought the dose down to very low, the stimulation of the lymphocytes was dramatic. And at that point, it suppressed the cancers in one case from 97 percent to 50 percent and in another 75 percent to 25 percent. The whole stimulation process was changed.

In 1921, with this group, there was a paper that I gave to Carmel Mothersill that she recently recognized in an article that said that they were looking at the fact that putting the serum effect, transferring the effect of the bystander effects through serum was done in 1921.

DR. LE GUEN: Thank you for your comments. You know, one of my old professors, 20 years ago, was Georges Mettier in Paris. So I was in Paris, France.

I was a big shift. And the other one was Georges Mettier. And one of the approaches of Georges Mettier was the immune surveillance concern.

But during the '60's, there was no tool. And now with all the new tools, we would have the opportunity to see if those hypotheses are not the new hypotheses. It was during the '60s that it will be possible or not to demonstrate this effect.

Thank you.

CHAIRMAN RYAN: Yes, sir?

DR. WILLIAMS: Alexander Williams. I work for the Department of Energy.

One of the theories in this country that has been used for regulatory purposes is the whole concept of collective dose. And there are some specific instances where this has been carried to lengths that border on the absurd.

For example, I remember some former colleagues of mine at the Environmental Protection Agency who believed that krypton-85 releases during nuclear fuel reprocessing would be distributed in the atmosphere throughout the world and would, consequently, provide a radiation source to everyone in the world.

So you could take the very small doses

from the krypton-85 and multiply by the population of the world and that this could be used for estimating some health effect. That was one of the more absurd uses of this whole concept.

Now, in terms of nuclear waste disposal, the department does regulate certain nuclear waste disposal facilities. The Nuclear Regulatory Commission does regulate it. And the EPA also has a role.

I won't take up everyone's time by going in who does what, but we are seeing situations where relatively small doses are hypothetical doses, are being attributed to individual recipients, sometimes over a number of people, sometimes in the distant future, the distant future from assuming that something in a nuclear waste facility migrates through groundwater and sometime in the distant future gets to somebody.

Given your presentation, it would appear to me that you're not a true believer in this whole idea of taking small doses and multiplying by lots of people and claiming that this is science.

So I thought I would ask for you to comment on the whole idea of population dose, where are the limits to that, what makes sense in your view,

what does not because, at least here, we do track facilities by the dose to the workers occupationally. This has an unfortunate drawback because it includes workers at a facility who are actually working in radiation areas and workers who are not, clerical workers, security staff, whatever.

So could you perhaps elaborate somewhat on that as to what your views are, what is reasonable in your opinion, what is not? I see some things here that are absurd, but perhaps there is something here of value. What do you think, sir?

CHAIRMAN RYAN: I think just as an introductory comment, I would mention that the ACNW has commented on collective dose in a couple of different fronts. And I think if I heard Dr. Le Guen this morning talk about it, you started with the idea that collective dose from a risk assessment standpoint was not effective.

And, again, just from our own comments, we have identified one good use of it. And that good use of it is in worker dose planning. For example, if we want to take out a steam generator or do an activity that involves ten workers and individual doses, it's a tool.

DR. LE GUEN: Yes. It's a tool.

CHAIRMAN RYAN: Not to assess the risk but to assess, can I do a better job? And I think it is a reasonable goal, although I might have others that would disagree, that if we can keep doses lower, that's not a bad thing.

So in the ALARA context of evaluating process one versus process two to accomplish a task, I think that is one where I would certainly personally think that is a reasonable use of it.

But the micro dose to mega people I think is at the extreme of where if you're using that as a risk assessment, that's off base. I'll give you my simple-minded example of my own.

DR. LE GUEN: Yes.

CHAIRMAN RYAN: Which would you rather be hit in the face by:@a 200-mile-an-hour wind for one hour or a one-mile-an-hour wind for 200 hours? The same amount of air is going to go past you.

I think that is the kind of extreme that takes us away from an effective us of collective dose.

DR. LE GUEN: And ICRP changed a lot in this field. And today this is also the ICRP approach to say, "Well, collective dose is a tool. It is interesting to compare from one plant to another, but it is only a tool to manage but don't use this

approach if you want to assess a risk. It will be a mistake."

CHAIRMAN RYAN: I think we have written letters on that topic. And that view that you just expressed is very consistent with our previous advice to the Commission.

Any other questions or comments? Yes, sir?

MR. REEVES: My name is Glenn Reeves. I contract for the Department of Defense.

I'm just wondering. For nuclear workers, three-fourths of their time is actually spent off the job at background or radiation. Does it make a difference at low doses for multiple chronic exposures versus continual radiation?

DR. LE GUEN: Because we are close to the natural radiation, in fact, it is very hard to answer your question. In fact, it is not to continuous exposure. You know, the really continuous exposure doesn't exist. One day you take a small dose. Afterwards you have nothing during a few weeks. After you will have a new one.

And so chronic, real chronic, exposure doesn't really exist. But it is difficult to answer your question because, in fact, you have a much more

real, continuous exposure with the natural background with the nuclear workers today at this level. That's true.

MR. REEVES: I guess one of the things that prompted the question was supposing you did have a fallout field where, of course, there was a gradient.

DR. LE GUEN: Yes.

MR. REEVES: How long should you spend in which areas? And would this make a difference?

CHAIRMAN RYAN: You know, that's an interesting question. Maybe I can ask you to shape it a little more tightly. I would think that it would depend on whether you were talking about responding to it initially, dealing with it in terms of like a clean-up type of situation, or what residual you would be satisfied leaving behind.

I guess I would see those as three different questions. Would you agree with that?

MR. REEVES: Yes. And that was the whole point of it. Would repeated exposures make a difference as compared with having someone chronically returned to the area to live.

DR. LE GUEN: You know, today I can give just an example, EDF. We are thinking about the

follow-up of people once they are retired. And we have the possibility to continue to follow those people.

And one of the goals is to assess the real risk at EDF. And for the moment, we have never observed an increase of cancer risk due to ionizing radiation.

There is this famous L. C. Walker effect that we saw at EDF. So there is no risk due to the exposure. And you know why the L. C. Walker effect is due, probably because we follow so much those people that we can very easily detect early if there is cancer or not. So the mortality is less important than the world population, the French population.

CHAIRMAN RYAN: Thank you.

DR. MUCKERHEIDE: Just a brief comment on worker doses. As pointed out, worker doses are going down a great deal, especially in nuclear power plant kinds of contexts, a little less so for nuclear medicine but, even so, they're going down quite a bit.

It's really more difficult to believe that we're going to get good assessments of worker dose effects as we don't keep track of nuclear medicine procedures, natural background, et cetera, which are enormously affecting who is getting what dose. The

guy with the lowest dose in --

DR. LE GUEN: Absolutely. I fully agree.

DR. MUCKERHEIDE: It's one nuclear medicine procedure. And he's got the highest dose of the group.

DR. LE GUEN: Yes, yes, yes.

CHAIRMAN RYAN: Don't wear your badge to the doctor's office. Yes. I appreciate that point.

One other area or study that we have not touched on I think everybody is aware of is the studies that are going on in the populations from and around Mayak in the former Soviet Union, where the chronic doses are relatively high, where the plutonium exposures are relatively high.

DR. LE GUEN: Yes. Mayak, Mayak, yes.

CHAIRMAN RYAN: And we now have a cohort of folks who have received, relatively speaking, much higher doses for more extended periods of time.

DR. LE GUEN: And you know that they have observed a threshold at Mayak.

CHAIRMAN RYAN: I'm sorry?

DR. LE GUEN: They have observed a threshold at Mayak. Yes. In the case of internal exposure, there is a real threshold.

CHAIRMAN RYAN: So there is an opportunity

there to follow those studies as those papers become available and get published and peer-reviewed. We will see how that goes.

With that, if there are no final last questions, I appreciate everybody's participation today. We have had a broad range of participants, a board range of views.

And I want to most especially thank our French colleagues for so expertly sharing their time, talent, and work with us today and thank everybody who has participated. I appreciate the opportunity to bring this to the record for the ACNW. It's very helpful to us. And I hope it's been informative for all of the participants.

So, with that, we will adjourn for our lunch period. And we will be back at 1:30 to bring up on the topic of a white paper on potential advance fuel cycles with Allen Croff leading that discussion.

Again, thank you all very much. I appreciate your time and participation.

(Whereupon, a luncheon recess was taken at 11:55 a.m.)

CHAIRMAN RYAN: I will reconvene our afternoon session, and I'll promptly turn it over to our cognizant member, Allen Croff. Allen.

VICE CHAIR CROFF: Thank you. This afternoon we're going to hear from a team of consultants, and you, John Flack, is being a consultant in this context, about the White Paper on spent nuclear fuel reprocessing, and refabrication, which we'll call recycle. This is going to be a verbal report on a written White Paper that's in preparation.

I think before going on, I guess we've got somebody on a telephone link. Could you introduce yourself.

MR. SEEHAN: Yes. Hi, my name is Daniel Seehan. I'm with the U.S. Government Accountability Office. I'm in Denver.

VICE CHAIR CROFF: Okay. Thank you.

MR. SEEHAN: Thank you.

VICE CHAIR CROFF: It would probably be useful if you'd mute your phone out there. We'll have some questions later on, but for now, to keep the background noise down.

MR. SEEHAN: I will do that. Thank you.

VICE CHAIR CROFF: Okay. Thanks. With that, we're sort of going to do a tag-team kind of a thing here. And, Ray, are you going to run the show? Okay. Our first speaker is going to be John Flack.

He's going to sort of tell us what the impetus for this whole thing was. John.

MR. FLACK: Okay. Thanks, Allen. First let me - we should have an agenda here - here we go. Let me just quickly go through the agenda, and we'll be talking throughout about the purpose of the White Paper, and the role it serves in supporting the committee's activity in response to the commission SRM. I'll start off with some brief introductions on that, which will be followed by Dr. Ray Wymer, former ACNW member, and then Lawrence Tavlarides from Syracuse, the Department of Biomedical and Chemical Engineering, will cover the flow sheets and the UREX+1a process. And then Ray will come back and talk about plant design of facilities, and that work was actually supported by Howard Larson who is to the right of me. Everyone knows Howard from ACNW. And then I'll talk about - following that presentation, I'll talk about the regulatory connection to all this, and then we'll leave it open for discussions of issues, and so on.

Okay. So to begin, the work itself actually stemmed from an SRM from the commission that was written earlier this year in February, and the commission was interested in the committee staying

abreast of reprocessing of spent fuel, and they should be ready to provide advice to the commission, as needed. And one of the important areas that they wanted the committee to focus on was the decommissioning, and, of course, the decommissioning is part of that process.

So at the time, we had this item as a Tier 2 item in our action plan, and it still remains a Tier 2 item in the action plan, but the commission thought that should reprocessing, new approaches to reprocessing evolve, that we may want to consider moving it, the committee may want to consider moving that to Tier 1. So we went back to the action plan, as you remember, and we revised it to really do three, and incorporated three things, which you'll hear about today. First, is that the committee become familiar with the fuel cycle for the advanced reactor systems, and that's pretty much the objective of the White Paper, is to bring out that information, to go through it from a historical perspective, and Ray will get into this a little bit more, and familiarize the committee through the use of that process with these new systems.

It's also the purpose of the White Paper, as well, as in response to the action plan, is to keep

informed of new issues, technical issues, regulatory issues as they evolve. And then, finally, in 2007, we had in the plan that we would do a site visit, and that's in the works now, as we're planning a trip to France to visit their reprocessing facility.

Okay. Just before turning it over to Ray, let me just mention that things are somewhat in a state of flux. We have -- well, let me go back just one view graph and just remind the committee of what has been done to-date, so far. We had several meetings. We had meetings with the staff in June, with DOE in July, and then we will meet with the staff again next month, and we'll hear the latest on their plans. And things are evolving in some extent with respect to DOE, and so, in this sense, we're really doing the second bullet there, keeping the committee informed of all the technical developments.

With respect to DOE now, when they first came in in July, they were talking about building a demonstration facility, which would be like a smaller scale of what would be envisioned to be a commercial production facility at some time. When we had visited Idaho this past month, they indicated they were no longer going to pursue that path, but they were going to go to full commercial scale operation. However, we

may find some additional information, and Ray may want to share that with you, that they may have gone back to reconsidering the demonstration facility. Clearly, if they do not build a demonstration facility and rely strictly on the engineering scale demonstration, there will be a substantial gap between what can be demonstrated on an engineering scale, and the full scale commercial production. So they're moving along right now with trying to get together an RFP for the commercial scale consolidated fuel treatment center, which is the third bullet there, and they're hoping to get out an RFP by the end of this coming fiscal year. And so that's clearly high on their priority list right now.

And in light of that, there would be planning on, if the schedule was to flow as they're envisioning it, they would be coming in with a license application December 2008.

And then, finally, there's the advanced burner reactor, which is following a few years behind in licensing space of the consolidated fuel treatment center. And, again, they have made a decision on that, and they are deciding to go with a 1,000 megawatt electric -- well, let me correct that - just a 1,000 megawatt thermal, I believe it is, 800 to

1,000 megawatt thermal reactor, and that would be a sodium cool fast reactor to act as a burner for the transuranic waste coming out of the consolidated fuel treatment center.

So that's, again, these dates. The reason why I hadn't written any of these dates down on a separate chart is because they're probably changing as I'm speaking here, but that sort of gives you a feel for all that.

Okay. If there's no further questions, why don't I just -- well, we'll save to the questions to the end. Right? I think that was -- we'll just turn it over to Ray Wymer now. Dr. Wymer.

DR. WYMER: First, can everybody hear me? If you can't I'll turn it off.

(Laughter.)

MR. FLACK: You want the pictures, too.

DR. WYMER: Yes, I apologize. Okay. Well, let's go on to the next one then, John. The content of the White Paper, which will be out in a couple of months, discusses the historic experience of reprocessing, several of the international fuel cycle initiatives, the DOE recycle programs and flow sheets, which you'll hear from Larry Tavlarides, and then some of the design and operational features, which are

based largely on the Barnwell plant that nobody in the room knows more about than Howard Larson.

What I want to do today is give you a sneak preview of what will be in the White Paper, so you have an idea what's coming on. There'll also be a section, and you'll hear about this today, too - technical safety license and regulatory issues, that'll be John Flack's. And some discussion about approaches for ensuring operational safety, and then the path forward that we expect that DOE will be taking.

First, some of you probably know all this already. It isn't as though reprocessing were something new in the United States. We've had very large reprocessing plants at Hanford, Savannah River, Idaho Falls, Hanford and Savannah River, of course, the reactors were run to produce Plutonium, very low burn-up of the fuel, only a couple of thousand megawatt days per ton, instead of 30, 40, 0r 50,000 megawatt days per ton burn-up, which we have in commercial reactors. The low burn-up is to produce a high grade of weapons-grade Plutonium, and we've had three stabs in this country at commercial spent fuel reprocessing.

The West Valley Plant was very early. It

operated a while, but there were a number of issues with it that had to be corrected, and it would have been too expensive to correct all those, so they just shut it down and decommissioned it. Here's the plant that Howard Larson was involved with, Allied General Nuclear Fuels, which is sometimes called the Barnwell Nuclear Fuel Service Plant, and then the GE Morris plant in Illinois, which also never operative. It was designed poorly. The Barnwell plant was designed properly, but the decision by Carter to not proceed with reprocessing effectively cut the legs off of that one. The next one.

Well, while we've been stagnating, the rest of the world has not, and France is leading the pack on reprocessing in the world, and selling a lot of their technology. The UK, of course, is reprocessing. Both France and UK are doing total reprocessing, that is, they're reprocessing other nation's fuels at a cost, at a price. And Russia has been reproducing both some of their power producing reactor fuels, as well as a lot of the Plutonium production fuels. And Japan has had a small plant for a number of years. I'll talk about that more. China has a plant, and India, also, is a player. Next.

In a little more detail, these are the

types of fuel that these plants are processing as commercial still, they call it; 1,700 metric tons per year is a very large plant. This is more typical, and then the Russians have the Mayak plant, which is available for processing power reactor fuel. Japan has the Tokai plant, which is a very small plant, been running for a number of years. They're just bringing on line the Rokkasho plant, 800 metric tons of heavy metal per year, for a total LWR reprocessing capacity for commercial fuel of 3,814 metric tons a year. There are other kinds of reactor fuels that are being processed that are not LWR fuels, they're heavy water reactor fuels, for the most part. Sellafield in the UK is reprocessing some of the gas cool reactor and some MOX fuel, and India has some heavy water moderated reactor fuels they're reprocessing, for a total civil capacity in the world of 5,589. That's to be compared with the DOE current plan of building a 2,500 metric ton per year plant, a single plant which is about half the size of all the plants combined to this point. Next slide.

Well, in order to bring reprocessing back under the screen, there's a strong sentiment that you can't just go ahead with the old style process where people think that has a proliferation potential,

because the Plutonium is isolated as a separate and pure stream, which then is, in principle, available for making nuclear weapons. So the idea is build proliferation resistant fuel cycles, and there are several international initiatives to do this.

I provided you all with the International Fuel Cycle Evaluation Study that ran for about three years back in the late 70s. If you look at the current plans, you'll see that this is the grandfather. Almost everything that's being considered currently that's being touted as new ideas, it's all here, and this just never got off the ground.

Right now, the DOE is pushing aggressively for the U.S. Global Nuclear Energy Partnership, which I'll talk about, and Russia has a parallel program called the Global Nuclear Infrastructure. Next slide.

Well, INFCE, the study back in the late 70s, had the following parts; nuclear fuel cycle assessment; that is, what are all the fuel cycles. How could you make Plutonium available to developing nations for use in fuels without making Plutonium available to them for weapons production. It dealt with spent nuclear fuel storage, which, of course, is a current hot potato. It talked about improved nuclear safeguards, and then they talked alternatives

to Plutonium, and high enriched Uranium economy, one of them was the Uranium-233 Thorium fuel cycle. Next.

The Global Nuclear Energy Partnership, os GNEP, as they call it, has these following goals. First, expand domestic use of nuclear power, get rid of the major reliance on the Middle East now, and in the future, for providing oil as their major energy source, demonstrate a proliferation resistant fuel cycle. Larry Tavlarides will talk some about that later. Minimize the nuclear waste accumulation. And if I had to say what is the most important issue here as far as Department of Energy is concerned, it's this one. They dearly do not want to build another Yucca Mountain. And by following through on this GNEP proposal, they can, in principle, extend the Yucca Mountain repository. And if you do what's proposed here, then the feeling is that the Yucca Mountain repository can retain the fuel up through the year 2100.

Well, part of this scheme is to develop and demonstrate advanced burner reactors, because one way to accomplish bullet 3, is by doing bullet 4, separate out the actinide elements, Plutonium, Americium, Curium, and burn those in a fast burner reactor, and turn them into fission products, rather

than actinides, thereby removing the major heat source from what's stored in the repository, and allows you to store the fuel a lot closer together, so you can extend the lifetime of the repository, so this is a key part of the GNEP proposal.

In this system, you would work with other countries and establish a lease and return fuel cycle; that is, the other countries would lease the fuel from the United States, and then when it was burned up, they'd return it to us and pay for some of the fuel recycling.

Another feature of it is to demonstrate smaller scale reactors. Now the standard reactor size got to be about 1,000, 1,100, even 1,200 megawatt days per ton, I'm sorry, megawatts - megawatts electric. I'll get it, and these are very large reactors, and not all areas around the world necessarily need to produce that much power in one spot, so the idea is to develop better small reactors that could be distributed around, at a size that's needed in a particular area.

DR. WEINER: Excuse me, Ray. Is this intended for countries that do not have reactors now?

DR. WYMER: It's intended for any country that has them now, or will have them, who want to

participate in these kinds of service, now and in the future.

DR. WEINER: Okay. But it is not --

VICE CHAIR CROFF: Ruth, I'd like to hold the questions until the end, if we can.

DR. WEINER: Oh, okay.

VICE CHAIR CROFF: Because this one is really tightly wrapped.

DR. WYMER: Yes, that's why I'm rushing here. We really have very little time to get through what we have to present.

Anyway, the idea is to demonstrate improved small reactors. And finally, to design safeguards into facilities, like the reprocessing facilities, and reactors to make them more proliferation-resistant than they currently are. So those are the GNEP principal goals. Let's go to the next one.

This will be the Russian initiative. It's almost a carbon copy of the GNEP proposal, totally, independently initiated by Putin and Russia. They would establish the same kind of full service, they call it full service international nuclear centers, where they would provide not only reprocessing, but enrichment and fuel fabrication, full service centers.

And they would build these centers, not only in Russia, but in nuclear weapon states, any of the countries that you saw in the previous slide that have reprocessing plants would be candidates for participating in this program. And they're ahead of the United States in that they have already designated a pilot enrichment center that would be part of this global nuclear infrastructure in Siberia under IAEA supervision, and they would build a shareholding structure for countries involved in the centers so that the participating countries would be shareholders in the business. But in order to do this, there has to be some legislation passed in Russia to make this possible. Next slide.

Well, sort of an overarching program is what's called the Generation IV Initiative. There was a forum held in May of 2001, and the goal of this Generation IV Forum was to talk about new generation nuclear energy systems; in particular, new reactors. And they were talking about five of them, they identified five that they work on. PWR and BWR would not be brand new, but they would be better from the point of view of proliferation-resistant, and with respect to burn-up then the current Generation, so that's evolutionary developments, rather than

revolutionary developments.

Then, of course, they want to continue to develop the Fast Burner Reactors, both the LMFER liquid metal, which could be either sodium, or bismuth, or NAC, sodium potassium, or lead, even, and gas cooled, generally speaking, helium cooled fast burners. Then the fourth type is the High Temperature-Gas Cooled reactor, of which there are two kinds; the German version, which is a pebble bed reactor, I'll say more about that, and then U.S. version, which General Atomic built and operated out at Fort St. Vrain outside of Denver for about a decade, which is built based on a prismatic fuel block. And, finally, the final one is the molten salt reactor, which is a radically different design from any of the above, in that the fuel is a fluid. It's a molten salt that is circulated through a heat exchanger, and it's Oak Ridge Development, which was shelved a number of years ago. Next one.

Well, if you want to talk about reprocessing and stick to light water reactor fuels, which is all there is at the present, well, light water, heavy water reactors, they're all there are at the present time. The current process is the Purex process, which some people believe have some

proliferation risks because it does isolate Plutonium as a pure stream. And that's the only process that's practiced on a large scale throughout the world. And there's a great deal of experience with the Purex process. However, there are proposals, and the U.S. proposals are contained in what I've called the UREX Alternatives, uranium process, and a French process called the Ganex process. Now let's take a look at the Purex process.

This is a grossly simplified view of the Purex process, but it gives you the essential steps. Irradiated fuel is brought onto the reprocessing site. It's chopped up, at which point some gases, like Tritium, Krypton, perhaps some Iodine come off, and then it's dissolved in Nitric Acid. You get some more off gases, you get some more Iodine off here, and then it is treated by a solvent extraction process, where you mix up the solution of everything, Uranium, Plutonium, fission products, Americium, Curium, everything, in Nitric Acid. You shake that up with Tributyl Phosphate, which is an organic solvent which is immiscible with aqueous solutions, and the Uranium and Plutonium preferentially are extracted into the Tributyl Phosphate phase, the fission products are left behind in the aqueous phase, and the Nitric Acid

phase. They become a high-level waste. The Uranium and Plutonium are separated in another subsequent step here. This is why the Purex process is considered to have potential for proliferation, because it isolated a pure Plutonium stream separate from the Uranium stream. And then the Uranium is further purified, making a Plutonium Oxide product. The Uranium is purified, as well, and can be re-enriched, and recycled, if you like. The Plutonium Oxide can be mixed with Uranium Oxide to make what's known as MOX fuel, or Mixed Oxide Fuel, which part of the highly enriched Uranium is replaced by Plutonium, thereby reducing the need for mining and milling more Uranium. Next slide.

Now these are the UREX alternatives that were considered by the Department of Energy, and several advisory groups that they assembled. This is the one that they settled on, the UREX+1a, and that's the one that Dr. Tavlarides will be discussing. Here, you get the following separated product streams, Uranium as a pure stream, Technetium as a pure stream, Cesium and Strontium together, all the transuranic elements, and all the other fission products.

This is the stream that's put into the fast breeder or fast burner reactor in order to

convert all those fuel elements into fission products, which then have a relatively short half-life, and are not nearly as heavy heat producers in the long term as the true elements are. Cesium and Strontium in this scheme are separated, because they both have about a 30-year half-life, and by separating those out, you remove also a lot of heat in the short term, and you can just set those aside, and after 300 years, they've decayed 10 half-lives down, which means they're at 1/1000th of the concentration that they were originally, and become a low-level waste.

Technetium is separated out separately because it's such a troublesome isotope in waste disposal, and it bogs Protectataydyne which is very mobile in the environment, and turns out to be one of the long-term products, long-term problems in a repository. So that's the UREX+1a process. Next.

Now, the French have independently come up with a process which they call the Ganex Process, called COEX, a co-extraction process, where they dissolve the spent fuel. Of course, they have off-gas streams there and there, and then they do an extraction and take out the actinides and lantonide elements. And then they strip out the actinides, which then they can burn. This is a simplified flow

sheet, and they shove out the lantonides, which become a waste, and they strip off the fission products, which become a waste. The actinides are recycled back, and in our conception, they are put into the fast burner reactor. But you notice they do not take out the Technetium separately, and they do not take out the Cesium, Strontium separately. They are planning to introduce this into their major reprocessing plant at La Hague around the year 2040. This will replace the PUREX process in their present plant. Okay, next.

MR. FLACK: Okay. I think this is --

DR. WYMER: Right. Now this is -- Dr. Tavlarides will give the presentation on the Urex flow sheet equipment. Larry has been scurrying around. They had a special meeting for him at a mixing symposium, and he was honored by a session in his name, and that was this past weekend, so we're lucky he's here.

(Laughter.)

DR. WYMER: I'm not sure he's awake, but he's here.

MR. TAVLARIDES: Well, you'll find out if I'm awake or not by what I say.

MR. FLACK: That was very fast.

DR. WYMER: You can ask questions.

MR. FLACK: Later.

DR. WYMER: Later.

(Laughter.)

MR. TAVLARIDES: Thank you, Ray. Thanks for the introduction. I flew in from San Francisco last night to Syracuse, and I got home about 11:30, and then got up at 4:15 this morning to get here, so it's been an interesting day so far. Well, anyhow, I'm happy to be here and speak about the work we're doing and these flow sheets that we've looked at and developed, so if I can have the next slide.

This gives you the basis of the flow sheets, and what we wanted to do is, amongst other things, determine the compositions of the process streams and the waste products, the effluents, and the other effluents that you get from the process, so that we know what their compositions in curie levels are so we could decide whether or not they are going to create problems for workers, as well as public problems, as far as the radiation being distributed and coming out of the process. So in order to do this, we had to get information about the nature of the radioisotopes in the processes, and to do this, we had looked at - we want to look at four cases. There

are four cases studied to evaluate for this UREX+1a process is 45 gigawatts per day per metric ton of initial heavy metal, and we're going to look at that for two cool down periods, one at five years, and one at 30 years. And the cool down ponds in the second is 60 gigawatts per day of metric ton of initial heavy metal, five and 30-year cool down time. The process sheets will be run at one metric ton of heavy metal per day, which is an engineering scale limit, and this can be expanded and scaled up if we want to have the two masses of all the waste streams and products that are being produced, and what their radiation levels are.

The flow sheet analysis preparation was done for us at Oak Ridge National Laboratories, and we used the ORIGEN burn-up code to make the calculations. And these were done for us through these gentlemen, Dr. Ruston, Guald, and Murphy. And they created all this information. It's now in the hands of the folks at Argonne National Laboratories, and they're going to run the AMUSE codes for us to give us the process streams compositions for these four different conditions.

To give you an idea, a typical power high pressure water reactor assembly has the following

breakdown of the fuel rods, and the heavy metals in it are Uranium metals, about 461 Kgs, now Uranium Oxide is 523, Zircaloy for the cladding and guide tubes about 108 Kgs, stainless steel end fittings, implant fillings, and Inconel and nicroblaze alloy, giving you a total hardware of 134.5 Kgs, along with the Uranium metal or Uranium Oxide. So that's the material that you're starting with. Can I have the next slide, please.

To give you an idea what these look like, this is a typical fuel power pressure water reactor fuel assembly. It has head end and bottom end assemblies which hold the tubes into place. And the tubes that are going to be processed look such as this. You have the Uranium elements, pellets in it, springs holding them in place, and there's space above and below it, so that you have volume for gases to be evolved and retained in it. These are sealed, and so whenever we try and process them, we want to chop these fuels up, these fuel rods out, gases are liberated, and you can access the Uranium and dissolve it out of the tubes, and out of the hull cladding. So the next slide then shows you a process scheme of the whole situation.

This is an overall view of what happens,

and I colored it in three color tones. The light blue is what we call the head end unit operations, the gold colors are the central operations, and these are the four separation processes that take place in UREX+1a, and the purple are what we call the tail-end process. I'd like to go through these with you, so you have an understanding of what is involved.

If we look at these fuel rods that we mentioned, they come in as spent fuel, and you see these rods and the assembly. These are chopped, and there's a chopped fuel assembly unit that chops these into different pieces. The hulls are placed into another process where they dissolve Uranium out of the hulls, and they create a Uranyl Nitrate solution. This goes into a clarifier to separate out the solution from any undissolved materials. This then goes into the main central unit operations, and we'll discuss that in a moment. And the stream H-5, is what we need to get from the ORIGEN code, as far as what the composition of the actinides and fission products are, for any given fuel that has been burned at a certain rate and cooled for a certain length of time.

As you look at these processes, though, whenever you chop the fuel, we saw this at Idaho National Labs, they're actually doing this in one of

the hot cells we visited. You hear a swish and gases come out. The gases that come out along with other products. You have Iodine-129, Krypton-85, Carbon-14, and Tritium, as well as other gases. They come over, and these are trapped and processed by a variety of ways. And then we can capture the Iodine and the other gases in different forms, and they could be placed at the high level form for greater than Class C forms, and so this is one of the products that we get.

The other part of the head end process is that you recover the end hardware. If we dissolve the fuel from the hull pieces, these are cleaned, and then these hulls also could be radioactive and have some products in them, fission products. These are cleaned in a way compacted, and packaged for high-level waste disposal. Furthermore, for any undissolved solids that come into here, and these can be also packaged, and I'll mention what happens with this later on.

As we go into the UREX process, into the UREX+1a, there are four stages I mentioned. In the UREX process, the first step separates Technetium from Uranium, and we have Uranium Nitrate solution. And the Uranium Nitrate solution can be denitrated and solidified, and it's packaged for storage, so you have

now recovered the Uranium, and it could be packaged for storage and future use. The Technetium is recovered, and it's reduced to a metal. Then the Technetium can be added to a melting furnace, where you add some of the clean hulls, form a melt, and this could be packaged as a high-level waste for disposal.

As we go on to the process, the next step is the CCD-PEG, this is Chlorinated Cobalt Dicarbolade with Polyethylene Glycol, and there's another system they're looking to use. This is a Bobcat Calic Sereem material. This processing step removes the Cesium and Strontium, and the Cesium and Strontium is steam reformed and formed into Aluminum Silicate, and this is packaged, as Ray mentioned earlier, for on-site storage, or storage for the order of about 300 years in bins that are kept cool so that it could decay away after that time, and be a suitable waste for future processing of storage.

The remaining materials that come out are transuranics and fission products, and these then go through two more steps. The Truex process removes fission products, but they don't remove the Lanthanide, fission products and transuranics. They do remove them in a separate stream. This goes into the Talspeak process. One set of other fission

products, not the Lanthanide fission products, are Calcine, and put into an immobilized high-level waste form.

In the Talspeak process, we can separate the transuranics, and with that we also separate them from the Lanthanide and fission products. Lanthanide fission products, which are also Calcine, and placed into a high-level form. The transuranics, Plutonium, Americium, Cerium, and Neptunium can then be blended with part of the Uranium to make a solution, calcine it and package it for advanced burner reactors. So this is how we can recover the actinides and blend it with Uranium for future use for advanced burner reactors. May I have the next slide.

This is to give you an idea of some of the data we're using to put into the AMUSE codes from which we will be able to track the compositions of all these trains. This is ORIGEN data, 60 gigawatt per day per metric ton of initial heavy metal, with a five year cool-down. We have the heavy metal at one metric ton, and these show you the composition of the elemental gases and the radiation level that they have for this one metric ton, the transuranics, Neptunium, Plutonium, Americium, Cerium. Now the next slide.

And we go on with the Cesium, Strontium,

Technetium, and all the rare earth. I'm not going to go into the details, but this is the kind of information we get from the ORIGEN code, which will be used in these process flow diagrams. May I have the next slide.

Now at this point, I'd like to take you through these different processes to give you our perception of how they are at this moment, or at least the key points that we think are streams that we wish to follow. So the flow sheets that you will see include operations for off-product recycle, solvent wash, and solvent recycle, as well. But before I do that, I wanted to familiarize you, if you haven't already seen these. This is a centrifugal contactor, and these are what people will use to do the solvent extraction separations. Centrifugal contactor has a spinning rotor. The aqueous feed comes in, the fresh solvent comes in, and it's emulsified into a liquid dispersion that then goes through the core of the contactor, where the centrifugal forces separate out the aqueous stream, and the organic stream, by coalescing the emulsion. The aqueous stream goes on on to the wall and passes out as a product, and the organic loaded solvent leaves in another stream.

These are connected in a sequence of maybe

20 or more of these contactors, and the next slide shows you a connection of 24, in this case, that were used for the Oak Ridge test - sorry, the Argonne National Lab test. And this may have an extraction section, a stripping section, and a washing section in it, where maybe 10 or so are used for extraction, 5 or 6 were stripped, and 5 or 6 were washed. And this is a concept that is used in these separations. May I have the next slide.

So this is the UREX one. You can flip back, John, to the blue slide where I showed all of the - that's it. Okay. So now what we're going to do is look at these four detailed flow sheets. I gave you an overview of the flow sheets, but there are a lot of interconnecting steps in each one of these four flow sheets, and I wanted to show you what is involved in these to a point, to give you an idea of what they look like. So could you go forward, now?

Okay. So this is that H-5 stream that goes into the UREX+1a process, the UREX cycle. This stream goes into this series of extractors that you saw, and in this case, the Uranium and Technetium are stripped from it, they scrub the stream, they take the loaded solvent which has Uranium, Technetium, and then this is taken out of the solvent, and it goes into

another unit operation called an ion exchange. And this ion exchange then separates the Technetium from the Uranium, and it gives you a product. And this product is this Technetium that we showed you before, and this Technetium is a Protecnotate, which then goes to the process of being reduced, and eventually made into the metal we spoke of. Similarly, this provides us the Uranium product, the Uranyl Nitrate solution, which then can be created into another package form that could be used later on as a mixing with the transuranics.

You can have off-spec material, if they don't work well, then we can recycle them in this case, and other streams that you get are spent solvent. At the end of the 200-day operation or whatever, you end up with spent solvent. This has to be treated as a waste, so this is something we're interested in, in determining how to treat that. And what leaves the process, in addition to the Uranyl Nitrate and the Technetium, is the raffinate, which contains the transuranics, as well as the actinide products. They go on to the CCD-PEG process. Can I have the next slide.

That second yellow block that we saw in the overall diagram, this comes off of the UREX

process. And in this case, we want to remove the Cesium and the Strontium. Again, a sequence of extractor contactors, and we extract the Cesium and Strontium from this. And the Cesium, Strontium then comes through and is stripped with these different solutions, which I won't go into. It's stripped. It provides us a product of Cesium and Strontium. This then goes to steam reforming, as a product that I mentioned to you a moment ago. And this, then, could be made into aluminum silicate product.

We also have coming out of here the raffinate. Now this is Cesium, Strontium-free material, and this raffinate contains the transuranics, plus the rare earth fission products, and other fission products. And this, then, goes on to the next stage of the Truex process. When you see this, this is the Truex process. It comes in from the CCD-PEG, and in this case we removed as raffinates non-lanthanide fission products. This goes to calcination. We then have the product which contains these transuranics and rare earths. This goes on to the next process.

Similar to the other ones, we have a spent solvent stream. We recycle it during the process, but at the end of the year of operation, we can treat

that. Off-spec material is recycled and treated. Can we have the next slide.

This shows you the connection of the Truex with the Talspeak. Now in this last processing step, we have the four transuranic elements, plus the rare earth or the Lanthanide fission products. And these come into this process. The solvent here is the MHDEHP. It extracts out the fission products. These are then stripped, and all these rare earths go into calcination, as we showed you earlier. The product that we get from this are the transuranics, and these are blended, as we mentioned earlier, with Uranium to produce the advanced burner reactor fuel. Again, off-spec material can be recycled, just as recycled to the Truex, we end up with solvent at the end of the processing cycle, which would be within the end of the year. This has to be treated, and this, then, concludes the overall details in a brief way. So we have a lot of interacting steps here, and in order that we know whether these streams, and what their products are, and what their compositions are for waste treatment, then we have to analyze these. And it depends on the nature of fuel that you put in at the very beginning, as to what you get at the end product. Okay? So we want to look at a broad range

of these so we have an idea of what we're dealing with. May I have the next slide.

So this gives you a summary, and I'll give you two summary forms. The first is the types of waste form and products that we get, and their disposition. The head end, those were the light blue boxes that we showed you, the very first detailed process setup, or high-level process set up. You end up with hardware, hull compacted, material, this disposition would be probably high-level waste, but maybe greater than Class C waste. We have undissolved solids that came from the fuel dissolution. This will probably be high-level waste. We have Iodine-129, and depending on the mode of processing it, you may end up with crystalline Iodine-129. It could be high-level waste, or it could be greater than Class C, or even low-level waste, but it depends what you want to do with it.

For head end, you have Krypton-85, as a compressed gas, and this also can be produced in a form that's packaged. You also have C-14. C-14 can be made into a caustic Calcium Carbonate, Sodium Carbonate that can be stored either for temporary decay, or even long-term for the C-14, as temporary. And Tritium, Tritium can be treated up front by a

vault oxidation process, or other process to form Tritiated water, if you want to capture it there, and there are off-gases given in all of these processing unit operations that produce Tritiated water. Perhaps this could be brought back, and even have isotope concentration method to concentrate Tritiated water. We will also have Technetium metallic waste as a high-level waste.

The central, we saw that we have Cesium, Strontium as a waste form produced by the steam reforming process, high-level waste cooling binsets, Truex or Talspeak gives us fission products, either a Zircaloy metal matrix or calcine high-level waste. All those spent solvents, we showed you there, at least a half a dozen of these, these could be incinerated. Vessel off-gases could be recycled through the head end treatment, if they're Tritium or other compounds. Off-gas control system for secondary waste, this might be a Class C waste product. And in the tail end, we have packaged Uranium, transuranic product. This is high-level waste storage for fuel.

So finally, this gives us a summary of the flow sheet attributes for regulatory consideration. We have various amounts and types of gaseous effluents that are being produced. We were trying to quantify

these and understand what they are, but we have various amounts and types of these gaseous effluents. We have various amounts and types of liquid waste, high-level waste for vitrification and fission products, low-active waste for cementation and drumming, solvents that might be incinerated.

The amounts and types of solid waste, this could be equipment. We showed you hardware from the fuel assemblies. We have resins from some separations that we showed you, and there could be greater than Class C waste, and new regulations may be needed for this.

Interim packaging and disposal, we showed you the Cesium-137, the Strontium-90, and interim package and storage of the actinides. So with that, I'll turn it over to Ray.

DR. WYMER: Everything on? Can you hear?

PARTICIPANT: I can hear fine.

(Laughter.)

DR. WYMER: Okay. What I'm going to show you now is all based on input from Howard Larson, who is the world's authority on the Barnwell plant. He was, at the time the Barnwell plant was under construction, the President and General Manager, and then most recently, many of you will recognize him as

having been a member of the senior staff as your team leader. I'm stealing your stuff, Howard. I hope you don't mind. Next slide.

None of this will be new to the people in here who have been involved in reactor licensing. They're very much the same considerations, except for proximity to reactors, of course, so I won't dwell on that. Let's have the next one.

The major facilities in a reprocessing plant, such as being envisioned in the Global Nuclear Energy Partnership initiative that DOE has underway, and the President of the Barnwell plant are fuel receiving interim storage for spent fuel, the separations process, which in this case was the Purex process, in the future would be one of these UREX processes. After the separations, the facility for Uranium product preparation, for Plutonium product preparation. This is what was done, not what would be done, because you would not have a Uranium product, Plutonium product preparation in a new reprocessing plant under the GNEP concept. Waste storage and solidification, high-level waste by vitrification. Next.

The routine releases that were considered at the time of the Barnwell plant were only those that

left the plant through the air. Everything else was packaged and managed in some way. It was not released to the environment directly. That's a major design consideration. All the process were made of corrosion-resistant equipment, by and large, stainless steel of one kind or another. There had to be confinement, and there would have to be in the future, against natural disasters, earthquakes, tornados, plane crashes, which is not exactly a natural disaster. All the high-radiation cells would be remotely maintained. There would be no direct maintenance.

Access to the various radiation zones in the plant are controlled by levels of radiation, each different level required a different set of rules, and a different set of management criteria. And, finally, criticality control has to be designed. Typically, this means keeping any equipment that has enriched Uranium, highly enriched Uranium, or Plutonium in it, either in a slab configuration, or in a tube that's no greater than four, five inches in diameter. Next.

Typical effluents, you just heard this from Larry, are the Krypton, which as soon as you dissolve off the fuel, the Krypton-85 is released. Krypton is a noble gas, of course, and it's chemically

unreactive, except under very extreme conditions. For all practical purposes, it's always a gas. And in the past, it has never been recovered. It's just been turned loose in reprocessing plants. In the future, it may or may not be allowed, probably would not. Iodine-129 come off the Nitrate Acid dissolver solution. In the past, it has been removed either by capture. As Larry indicated, either trapping it as Sodium Hydroxide solution, in which case it becomes Sodium Iodide, or passing it over solids that are impregnated with Silver Nitrate, so that you form a silver iodide fixed material, but it wasn't turned loose.

Carbon-14, of course, would be put into this Carbon Dioxide. Larry indicated that that would be removed as Calcium Carbonate, which we precipitate, and in the past, that has been turned loose. Tritium comes out two ways. It comes out either as a gas when you share the fuel. Goes in as a fission product, which they turn the fission product, it's about one in every thousand fission produces a Tritium atom, and so it comes off as a gas there, or what doesn't come off that way, is exchanged with hydrogen and water in the Nitric Acid solution, and becomes Tritiated water, HTL. And these are basically unresolved issues at the

moment for future reprocessing plants.

Solids, of course, are some are vitrified, some are stored as other kinds of solid forms that have low activity and intermediate level activity waste, and miscellaneous waste for solids and liquids. Those are the typical effluent streams, and those are the primary considerations for the Nuclear Regulatory Commission's interest. And it is those that we are trying to quantitatively pin down in the separations processes that Larry talked about. The amounts and types will be indicated from the flow sheet runs based on the AMUSE runs that Argonne is doing for us, under our direction, and we are specifying the conditions of burn-up and cooling, cases they are to look at. Next slide.

You have some additional solids and liquid waste, which there's no sense belaboring. High-level waste typically comes out as liquids, stored in tanks, and then this is certainly what was planned at the Barnwell plant, and would eventually be vitrified. Typically, you store it for four or five more years as liquid. While it is short-lived, radioisotopes decay solids to stable isotopes. Next.

As I said, the high-level waste would be borosilicate glass. This is pretty much accepted now

by everybody as being a good way to solidify waste, whether or not it's needed. People are happy with borosilicate glass. The English, and the French, and we have been producing borosilicate glass waste, and people have come to accept in. And while there are other kinds of disposal methods, typically people who are not initiated in the business, will not settle for anything other than borosilicate glass.

Other types of solid waste could be solidified in cement if they're low-level waste, and high-level waste will be stored at a geologic repository, like the proposed Yucca Mountain Repository. Other kinds of waste in the past have typically been stored in surface trenches. That's probably no longer acceptable. And here's a problem. Iodine-129 - nobody has come up with a good way to produce a very stable chemical form of Iodine-129.

I was in Russia a few years ago, and they were talking, the guys come up afterward and said we got some tons of Iodine-129. How do you people fix that stuff, anyway? So I said I don't know, we've got the same problem you've got. And there is no truly stable inert form, and it's something that needs attention, but it's a problem. Next.

One of the key things at a reprocessing

plant is the processing personnel. It's like a reactor, you almost have to be qualified to fly a Boeing 777 in order to run a reactor. The same thing is true running a reprocessing plant. These people have to be highly trained, and these are the kinds of operations that are conducted, and you need senior operators, and this is based on Howard Larson's input, that he found that people would take this training, and they couldn't pass the training course. They had to go back and take it again, and again. It took about a year, to a year and a half to train operators to run the reprocessing plants, a major problem. Next.

Part of any complete fuel cycle involves fuel fabrication. Typically, the light water reactor fuel is composed of highly enriched Uranium oxide pellets about half an inch or so in diameter, and in place of highly enriched Uranium, you can also use Plutonium as part of the fissile material. You clad it in Zircaloy, and you have Zircaloid or some stainless steel hardware, as you saw in the slide that we showed earlier.

In the case of fast burner fast breeder reactors, oxides have been what's been used in the past. Carbide is being used in India in small

reactors, and Nitride has been considered, and these are fabricated into pellets, and they're clad in stainless steel because you don't need a low neutron cross-section cladding in a fast reactor. Nitride is a problem because Nitrogen is a source of - is the principal source of Carbon-14. It captures the neutron, Hydrogen-13 captures the neutron, eventually becomes Carbon-14, so if you just use Nitrogen as it's present, that you're breathing at the moment, it would make too much Carbon-14, and so in order to have a Nitride, you probably have to do a Nitrogen isotope separation, and use a Nitrogen isotope, which does not form Carbon-14. It's not a difficult separation. Light elements typically are relatively easy to separate isotopically, but it would be a significant step.

High temperature-gas cooled reactor fuels are typically made of Carbides, or a mixture of Carbide and Oxygen, or of Oxide. And these are, for HTGRs, these fuels are made into tiny, tiny pellets, less than a millimeter in diameter. That is what is the equivalent of a Zircaloy clad fuel rod. It's a tiny, tiny pellet, a kernel of which is one of these chemical compounds. And then you coat that tiny little inner pellet which is maybe half a millimeter

in diameter with a pyrocarbon coating which is porous. That gives you a space for fission product gases to accumulate without bursting the pellet open, and that's the equivalent of the plenum space above and below the pellets in a fuel element, PWR fuel element. And then on top of that, the porous graphite, there is a silicon carbide coating. All this is building up to something that's no bigger than a millimeter in diameter all tolled. So that silicon carbide then is the containment vessel, nothing can get out of that.

And then finally, on top of that, there's a graphite coating to protect the silicon carbide. Obviously, that's not much fuel, so there are billions of those that have to be fabricated, but this has been done on a commercial scale. And three reactors, to my knowledge, have been run. One commercial park producing reactor is Fort St. Vrain, and two test reactors in Germany, a small one, and larger one, which was a prototype.

There are two different ways that you can treat these tiny little spheres. One is, you can put the little spheres into bigger spheres. You roll them up in sort of what we might call the dung beetle approach, where you roll these up and it's wrapped in a tar matrix, so they're little - it's like a plum

pudding, they're embedded in this tar matrix, and you graphitize that, and you've got a graphite sphere. Those spheres are then put in a big tank with a conical bottom, that's the reactor.

In the case of the Fort St. Vrain type, the little spheres are put into sticks of tar. Those are graphitized. Those are stuck down holes in a great big graphite block, so that's a large fuel element. These types of fuels pose very special and difficult reprocessing steps, mainly in the head, and getting rid of all that graphite. Next.

As far as fabricating the Plutonium oxide, Uranium oxide mixtures are concerned that can be used in light water reactors, either PWRs or BWRs, called MOX fuel, Mixed Oxide Fuel. Those are being fabricated, have been fabricated, how they're fabricated shown in this chart. And we, of course, are building down at the Savannah River plant our own little indigenous MOX plant, which maybe some people in this room have been involved in the licensing of. So, you see, there's a fair amount of experience in fabricating MOX fuel. Next.

MR. FLACK: I think that's me.

DR. WYMER: And now we come to what really is the heart of the presentation. It's John Flack's

presentation on the things that are near and dear to the hearts of people in the Nuclear Regulatory Commission, make the Regulatory Connection.

MR. FLACK: Yes, I was going to say, we had the French Connection this morning, so now we move on to the Regulatory Connection.

Okay. I mean, we could spend a lot of time talking about the regulations, and I don't know whether I should stand up or sit. Let me just sit here, because I think we probably need to go through it rather quickly, but in any case, as you could see, what I laid out on this viewgraph is a framework, is the framework that we use today to regulate various parts of what might be considered pieces of the consolidation facility that DOE is proposing. But what I did in this case was stand back and try to understand what were the high-level, the top level regulatory criteria, because once you know the top level regulatory criteria, then everything else follows. And from a list like this, various regulations, the top level regulatory criteria would be like Part 50 and Part 70, and Environmental Protection Part 51, because it's there where you set the doses and the limits, that then you have to comply with.

One thing I noticed in doing this, coming from reactor space, is that in the reactor side, there's something else called policy issues. And the policy issues aren't, per se, regulations in the sense that they have to be met by law, but it often dictates how one reviews a licensing application. And there's three significant, for light water reactors, policy statements that drive a lot of the decisions in the agency; the Safety Goal Policy Statement; the Advanced Reactor Policy Statement, which expects that the next generation of plants are going to be safer; as well as the Severe Accident Policy Statement for operating reactors, but these are policies that the commission has put out, that says this is what we expect.

When I look at the reprocessing area, there's not really a policy statement. It's really the regulations that are there, that we're expected to use. Now maybe at one point, the commission may want to come forth with a policy statement, but that's up to them whether they want to say something about making reprocessing facilities safer than previous facilities, or something like that. But right now, we're really dealing with the regulations as they're written on the books.

So looking for, actually, the top level

regulatory criteria in really the three major areas that the NRC regulates; it's basically licensing the facility, certainly, the safety and security aspect is significant, the effluents that were just described before, what would be allowed, and then waste, the types of waste that goes to the disposal. So you have, for example, in this context, you're having Part 50/Part 52/Part 70, if that becomes the case, in actually the reprocessing facility itself, and Part 20 is really setting these dose limits, that then you have to design your plant to meet.

The next bullet, of course, is the oversight of the operations, and that, of course, is making what you license the plant to do, the performance criteria, how you regulate its operation. And for reactors, of course, we have a whole process called the Regulatory Oversight Process, that does that. You would have to envision some similar kind of process for reprocessing facility. And, finally, decommissioning, and we heard a lot about that yesterday. And a lot of that thinking and thought should be able to be carried over to something like reprocessing.

Okay. Looking at one of the more significant regulations, of course, is Part 51, and

the Environmental Impact Statement, so when an applicant would come in for a license in order to construct and operate a reprocessing facility, it would have to submit an environmental report. And that report would have to comply with Table S-3. And S-3 is rather interesting because what it does, is tries to say here's all the disposition of all the radionuclides that would come out of a 1,000 megawatt electrical plant if it ran for one year. And the scenarios it chose in those tables, and where it partitions everything, depends on the fuel cycle being either once through or Uranium-only recycled. So right there, you'd have to revisit Table S-3, and say well, what does it mean in the context of reprocessing? So, certainly, that would have to be something that has to be revised.

Once the report comes in, the staff would do an assessment and, essentially, write an Environmental Impact Statement. Now for a reprocessing facility, of course, there's nothing specific for reprocessing, so what would the applicant do when it comes in and submits for an application an Environmental report? Well, there was what's known as GESMO - if I can go to that viewgraph right now - that was done some years ago, that had a generic impact

statement. Now what it looked at was, it made a few assumptions - it said, first of all, by the year 2000, we would have 507 plants running, which we don't have today, so it was quite an assumption back in those days. But it said that if we went to MOX, how would that change things with respect to its impact on the environment? And so what it looked at was the years 1975 to 2000, and said that we would go to, essentially, 507 nuclear plants operating by the year 2000, and we would look at different recycle alternatives. And so, in that report, that generic impact statement, they looked at three alternatives. They had looked at Uranium Plus Plutonium recycle, and actually, there's more to it than that, because they looked at whether it was delayed at some point, and the timing was important, so there was other alternatives. Basically, it's the Uranium/Plutonium recycle, you would recycle that material. Then you would have just Uranium recycled by itself, and then no recycle.

And what they looked at was okay, depending on what alternative you chose, how would that alternative impact the environment? And they looked at key factors, including the plant effluents that we were talking about earlier, how would that

change the waste generated, the occupational dose, and the non-occupational dose. So what did they find?

Actually, they found in the conclusion, which is in the bottom of the viewgraph, that there was no clear preferred path or specified for the fuel cycle option based on waste management alone. But they noticed that, of course, that the various options resulted in at least three areas significant differences in the curies released to the environment. And, basically, for the no recycle, which would be the straight-through once right to the mountain, so to speak, versus other recycle options, you had Tritium increasing by two orders of magnitude to the atmosphere, and Carbon-14 about a factor of three, but Krypton-85, approximately three orders of magnitude increase. And this would - for example, the Krypton would be running from millions of curies to billions of curies, basically, in that assessment, since at that point, in that time it was just being released to the atmosphere.

But interesting enough, there was not really any increase in the non-occupational dose. Well, there was an increase in the non-occupational dose which quadrupled basically for the foreign population, since what happened would be the enormous

number of people that would be exposed to very low doses of radiation from the Krypton, but there would actually be a decrease in the occupational dose, since there would be less mining, and mill tailings, and so on, so the real concern was this large increase in dose over large populations, basically. And that was really part of the findings, the key finding from the GESMO work that was done, and this is back in '76.

Shortly after, EPA released its standard, and this is what you might consider to be a top level regulatory criteria. They said that -- actually, in that standard they specified the levels of releases for the operation of the Uranium fuel cycle, which means over that fuel cycle, there should be certain, not doses, but amounts of curies released for Krypton, as well as - well, let me put it - it's actually on the next viewgraph you have. The Krypton-85, the Iodine-129, and the Plutonium and other millicuries that would be allowed to be released over the entire fuel cycle for a 1,000 megawatt electric plant. So this was one of the major outcomes of the standard, which sort of set the stage then for the entire fuel cycle.

Well, there were some major issues that were raised during those reviews, and three of them

were that the standards were unnecessarily conservative, since they were talking about collective dose, and that they disagreed over the need to control Krypton-85, and the relationship between the health effects and dose, and that was because of these very small doses over large population areas.

So, in any case, EPA set that standard, and there was, of course, two parts of that. One was the actual curies released, and the other one was the dose to the members of the public. And it said that for the cycle, again, the 1,000 megawatt electrical power per year, the whole body dose should be less than 25 rem, thyroid 75, and to any other organ, 25 millirem, sorry, millirem. So with that said, that sort of set the stage for the NRC regulations, which are contained now in Part 50, Appendix I, which is ALARA for the light water reactor effluents, which was actually talked about yesterday. And I think there was a question on Ruth, where do these numbers come from? Well, it's coming from that EPA standard, which then the NRC interpreted to mean for these various releases of liquid to be less than 3 millirem whole body, 10 millirem to any organ, and a gaseous effluents of 5 millirem whole body, and 15 millirem to the skin. And then, finally, for the radioactive

Iodine and radioactive material, less than 15 millirem to any organ.

Now the reasons why they're lower than 25 millirem is, for a number of reasons, but the main reason is for multiple units at a site. For example, you would have - this is per thousand megawatt electric, so it would need to be some fraction of that. And, again, if one was to build a reprocessing facility at a site with a plant, one would have to consider these doses to any member of the public.

CHAIRMAN RYAN: Just a quick point while we're here. These doses are cast in ICRP-2 annual doses frameworks, not the current doses, so we don't do organ doses, or thyroid doses any more. It's total effective dose equivalent, which is an integrated --

MR. FLACK: Oh, is that -- okay.

CHAIRMAN RYAN: So the numerical values here may or may not reflect what would be selected.

MR. FLACK: I see. Okay.

CHAIRMAN RYAN: So they're very, very different. They're actually based on dosimetry from 1959.

MR. FLACK: Yes, so - and that's --

CHAIRMAN RYAN: It's just enough.

MR. FLACK: Yes, right. This is what's in

the regulation today, so things need to be --

CHAIRMAN RYAN: Like 10 CFR 61, they're out of whack.

MR. FLACK: Yes, they need to be revisited. Okay. So that's -- well, this would be, then, the top level regulatory criteria, since this is what is being actually implemented out there right now by the NRC. Okay. So the next part of that, the next part of what regulations is covering that I wanted to talk about, is the licensing of the facility itself. And, basically, looking at where the regulations are today, there's really one of three options that one could use to license a facility, like a reprocessing facility. It's to modify the current regulations, come up with a new rule, or to use the ongoing effort in rule making to develop a technology neutral framework that could apply to this technology. So this one just mentions, basically, the three kinds of rules that are there now. Part 50 is generally used for licensing light water reactors, but it is the rule on the books right now that one would use to license a reprocessing facility. Part 52 is more of process-type rule that helps expedite the licensing of new nuclear power plants by combining the construction and operating license into one package. And then Part 70,

of course, is the one that is used to license special materials.

So looking at those that are on the books today, there are certain pros and cons in using each of these regulations. For Part 50, of course, there's a lot of experience in licensing space with using Part 50, but the con is it's primarily used for licensing light water reactors. I think what's important about Part 50 is the structure it presents and the way it processes the license in identifying, or the process really flows from no what accidents you want to protect against, what is the design criteria that you want to use to defend against those accidents, put in place, identify the systems, structures, and components that will be then monitored with oversight, understand what source terms would come out of these accidents that could occur at the plant, and then do a PRA to assure that you've covered everything, and if not, feed that back into the licensing process. So Part 50, although it doesn't require a PRA, per se, it does require the identification of events and accidents in the context of design-basis accidents, and licensing-basis accidents, which in today's space would rely strongly on a PRA. So even though it wouldn't require a PRA for licensing, it would be

surprising to have a plant go through the licensing process without one today. Everyone uses the PRA today for these kinds of things.

Part 70 - Part 70 has experience with fuel cycle facilities. They use what's known as an ISA to do that same kind of work, but it would require substantial revision and, in fact, a change in philosophy, the way they look at risk in that licensing process.

Well, let's move on, because there was a few comments made on that later on. The other options for licensing would be to develop a new rule. And, of course, the advantage is that you could make it very specific to reprocessing. The disadvantage, of course, is it would resource-intensive to develop a new rule. And, of course, the time may not fit in with the schedules that DOE is talking about in submitting the license application.

There is this other new framework that is going on under Part 53. The advantage, of course, is that it is in the development stage, and one could essentially go in there and how to accommodate a reprocessing facility, they would need to do things differently, maybe, with the way they're doing that work. But, again, it talks about working with the top

level regulatory criteria, and then from that implementing in the Reg Guides what it would take, as it would apply to specific designs or technology. Again, right now, this is only for reactors, so it would be something that would have to go into that process.

Okay. As I mentioned, there is a difference, rather significant difference, I think, and the committee had thought some years ago, about ISA and PRA. And, in fact, the ACNW wrote a letter on this in 2002, and challenged the staff on its decision to use ISA methods to risk-inform activities, rather than to employ PRA methods directly. And they questioned the effectiveness of ISA leading to desired outcomes. And, basically, what are those desired outcomes?

Well, those desired outcomes are really, again, to understand what kinds of events can occur at a plant, be able to defend against those kinds of events in some way, shape, or form using safety-related equipment, or equipment that would be under some category of surveillance. And then to understand what risk meant to the public, and make decisions on using that type of information.

There were some recommendations that came

out of that letter for the staff to move to a quantitative risk assessment. Basically, one of the things they commented on, the committee at that time, was that it didn't treat dependent failures, and those that are familiar with PRA know that that's a major contributor to risk, and the way things are modeled, and dependencies are treated. And, of course, getting back to the point that I was just making about the aggregated risk, or the full risk perspective, and being able to make decisions on that. And then, of course, the treatment of uncertainties. Uncertainties are a very important part of the PRA, and how you treat them in defense-in-depth and other ways is a very important aspect that is not being considered in other methods, such as ISA. Now you could, maybe, account for it in some way, but at this point, the way the PRA uses them, it's a very formal process, and a very important part of the PRA process.

Okay. One other part of the regulations actually did change because of West Valley, and that's Appendix F in Part 50. And that had to do with all the situation that evolved in West Valley in trying to decommission the plant, the facility. In fact, what were the numbers? Originally, it was estimated that to decommission West Valley would be $4 million, and

so far it's up to $2 billion, so the agency at one point felt that something needed to be done, and so they actually put in Appendix F to try to prevent things that have happened there, from occurring in the future. And, of course, one of them is being sensitive to the high-level waste issue, the liquid waste, and that limit it to five years, solidification of the waste, and transfer the waste to a federal repository within 10 years. And, also, the waste only being deposited on land owned and controlled by the federal government was added. And I thought the fourth bullet was much in line of what we talked about yesterday, which needs to be done now, and that is, that the design objectives also facilitate decommissioning. And then there's a question of the financial qualifications of one going into that business. So this is also an important part of the regulations that has been put in place that specifically address reprocessing.

Okay. Just to summarize some of the high-level areas for the committee to focus on as the agency goes forward in licensing, regulating, reprocessing facilities, the first, of course, is what licensing approach is the best approach. And if PRA becomes part of that process, then should there be

some safety goals associated with that, goals that the agency should try to meet, or have the licensee try to meet. And, although, safety goals aren't necessary - I mean, there are several international countries now that are using PRA in their process that do not have safety goals, and see the benefit of using the PRA alone in their decision making, is an important piece.

The other is the integration of the standards into the NRC regulations, and that goes back to what the EPA standard says today, and how that would be applied in the context of our regulations to reprocessing facilities. And this has to do with the issues that were discussed earlier about emissions, and so on.

And, finally, the design criteria for decommissioning, the guidance that would need to be developed for that, and what the expectations would be as far as the agency is concerned. So am I on time? I guess I'm running a little late, but I think that puts me back to you, Ray.

DR. WYMER: Well, thanks, John, that was very good. And I'm sure that that was what people came to hear. The ACNW member who is responsible for overseeing the consultants in preparing this presentation and the paper, White Paper, is Allen

Croff, and he's been very diligent keeping our nose to the grindstone with respect to what is the specific - people who know Allen know that that's what he's good at.

VICE CHAIR CROFF: I had a good teacher.

(Laughter.)

MR. WYMER: He said, "You have several objectives, but the real objective, first and foremost, is that you want to tell ACNW what things they ought to look at in order to prepare a letter to the Commission." There are other things, of course, that are provided -- a resource paper for the staff at large, or maybe you're not an expert in reprocessing one, or something about it.

But mainly this is -- you know, Allen has been helpful in producing this list. Fifty percent of the criticism that you have with it should be directed at Allen.

(Laughter.)

Well, these are some of the suggested issues for ACNW consideration. Under the -- I've broken them into technical and regulatory. The things that you've got to pay attention to in your letter and providing some guidance on how to deal with these things are managing the off-gases, the iodine tripton,

carbon-14 as it is present in carbon dioxide, and tritium as it is present in gas and in tritiated water.

And some of the issues that you need to consider are: what are the appropriate measures of risk involved with these things? What are the acceptable technologies? I've listed a couple things here. But these are embryonic. There are ways of stabilizing -- separating and stabilizing the noble gases -- krypton, xenon -- but they have not been put into large-scale practice, and the same thing is true of these two. In iodine, I mentioned there's a real problem.

What are you going to do about cesium and strontium? Are you going to just set it aside and wait for it to decay for 300 years or -- so it's an easy to manage problem? Or just what are you going to do? And how about the uranium? If you recycle it, what -- if you dispose of it, what do you do? How do you manage it?

Next.

So additional technical issues that we think that the ACNW might want to think about is there will be large volumes of some of this waste. There will be a large disposal cost. It'll be -- in

general, it's going to be a problem. I think one of the latent 800-pound gorillas waiting to be spawned is there's very large volumes of fairly low-level waste. You know, really not enough attention has been paid to it, in my opinion.

Then, there will be some very different kinds of waste. When you get into pyroprocessing, when you operate the fast breeder reactor or burner reactor, it operates using a totally different kind of system, not an aqueous system at all but a pure salt system.

And it produces wasteforms which have not been certified, have not been qualified, and which Argonne National Laboratory, who is the lead in this area, are more or less saying, "We know that, and we think we can get people to go along with these wasteforms as being acceptable and certifiable." They almost have to. Otherwise, it can't use their process.

And what are the issues related to safeguards? You need to pay some attention to that.

Next.

We're not telling you what to do. We're telling you things that you need to look at on the regulatory side of the house.

You have just heard a lot about this, of course, from John, but which ones could be used? And if you use it, what changes would be needed? Or do you want to discuss the advantages or disadvantages of going to new regulations, much as was done for the Yucca Mountain repository? You know, you just ginned up some whole new regulations to deal specifically with Yucca Mountain. Well, that same thing could be done with reprocessing.

And then, to what extent should there be deterministic, and to what extent risk-informed? There are two camps here, even within the NRC on, how far do you go from deterministic to risk-informed, and are you losing more than you're gaining in some cases by going to risk-informed? So that's an issue that needs to be addressed, we think.

And then, what are the impacts on other regulations? I've listed a couple here. Is the classification system adequate, or do you need a new one? These ought to be -- you ought to think about it.

And is there another one? Is there one more there? Yes. This whole issue is related to decommissioning. That's a -- that's kind of a new one, and you're getting into the province there of

telling the plant designers how to design their plant. And you can certainly regulate that, you can do that, but you've got to be very careful, because they will -- they will resist that, in my judgment, and it has to be done in collaboration with them.

So you get something that really is a good balance between what the regulating agencies think should be done and what the plant designers think can be done economically and reasonably in the way of designing their plants with respect to ease of decommissioning.

What kind of regulations do you want on effluent releases? And how do you balance the risk to cost or technology limitations? DOE's position at the present time is we'll tell you what can be done, and that's what you will approve, because you can only do what you can do. And that may be okay, provided what they can do is good enough. So that's something you need to spend some time with.

And I think that's all that I have. We're running a little bit behind here.

MR. FLACK: You just mentioned the simple ones, right?

MR. WYMER: Yes, I don't -- down in here to the son of GESMO. That's Allen's phraseology.

And, of course, you heard about this from John. I think that's all that we have.

MR. FLACK: Yes, I think that kind of wraps it up. So why don't I, at this point, turn it over to Allen.

VICE CHAIRMAN CROFF: Thanks. Great job, like drinking from a firehose, but you made it just about in the allotted time. I think we started a few minutes late here.

I'm now going to go to the questions, and I'd like to suggest we start by each Committee member taking up to 10 minutes and asking whatever you want to ask of whomever you want to ask. If we have a little bit more time at the end, then we'll go around again, or allow some follow up.

Jim?

MEMBER CLARKE: Thanks. Thank you. That was a very interesting presentation. I'm peddling as fast as I can as well.

Ray, you mentioned that one of the key drivers for GNEP, and I certainly agree with that, or it should be a key driver, is extending the lifetime of Yucca Mountain or anything that has the intent of Yucca Mountain, and that that would be done through the separation processes, and then using fuel again in

other kinds of reactors, fast reactors, and maybe using it again. I don't know how many times you can do this.

But given the importance of that, and the value of that, have there been any calculations -- and I guess you'd have to make some assumptions -- but what would -- and I guess you'd want to do it on a mass basis, so would a mass reduction be if what -- what goes to Yucca Mountain now or would go to Yucca Mountain now versus what would go if this were implemented and successful?

MR. WYMER: The estimates are at least a 10-fold increase in the storage capacity. Right now, it's at a total capacity of 70,000 metric tons of initial heavy metal, and, of course, 10 percent of that is DOE waste versus commercial waste. The horseback estimate is a 10-fold increase in the storage capacity of Yucca Mountain.

MEMBER CLARKE: And does that take into account all the waste, the high-level waste streams that would have to be vitrified as well?

MR. WYMER: Yes.

MEMBER CLARKE: It does, okay.

MR. WYMER: My understanding is it's all pretty --

MEMBER CLARKE: The regulation is based on mass, but --

MR. WYMER: -- pretty embryonic --

MEMBER CLARKE: -- there are volume considerations, too, I guess.

MR. WYMER: Yes. But anyway, it's a significant increase.

MEMBER CLARKE: And what assumptions, do you know?

MR. WYMER: I don't know, and I don't think that you can know it better than plus or minus a factor of two sitting here today, but that's --

MEMBER CLARKE: How many --

MR. WYMER: -- that's a number that I've seen, is --

MEMBER CLARKE: How many passes do you get at it, at something like this?

MR. WYMER: Well, if I can take just a second, there are several ways that we haven't even talked about here that you can deal with these issues. For example, by putting lightwater reactors in tandem with heavy water reactors, you can sort of get everything but the squeal out of the fuel.

And the South Koreans, in collaboration with the Canadians, has come up with what they call a

duping process whereby you burn up the fuel as far as you can in a lightwater reactor, then you chop open the fuel, you heat it up to about 400 degrees Centigrade. That causes the fuel to fragment. Volatile gases come off, which are high cross section things for the most part.

Then, you refabricate that fuel into a fuel that you put into a heavy water reactor, which is a more efficient burner. And that could be an intermediate step stuck in, you know, before you go to this reactor burner. So there are permutations and combinations that haven't even been discussed here, and have not been discussed much internationally, but which people think about.

So it's a hard question to answer, Jim.

MEMBER CLARKE: I understand. I understand.

MR. TAVLARIDES: Can I make a comment?

MR. WYMER: Yes.

MR. TAVLARIDES: I was just looking at this table that I gave you about the origin data for the 60 gigawatt per day per metric ton. And it's interesting, if you look at the amount of uranium that is --

MEMBER CLARKE: I'm sorry. Which slide is

that, Larry?

MR. TAVLARIDES: It's 25.

MEMBER CLARKE: 25?

MR. TAVLARIDES: So if you look at the uranium, right in the center, okay, this is -- if you have one metric ton of material, the uranium that, if you can recover it all, is 923,000 grams. So you have recovered about 93 percent. You had the plutonium, you've got about 93 percent of the mass of the fuel that is there that you can recover and put back in. So that's not going into the repository.

MR. WYMER: Right. Yes, most of it would be uranium in the current plan, yes.

MR. TAVLARIDES: Exactly.

MEMBER CLARKE: Okay. Thank you.

VICE CHAIRMAN CROFF: Jim, let me try and help that just a little bit. Right now, what you can put into the repository physically is limited by heat. I mean, you've got these tunnels and they're spaced well apart to get the heat out. After you take out all of the actinides and the cesium and the strontium, there is very little heat left, so you can really pack it in. And it's just much closer together.

MEMBER CLARKE: That's a good point, Allen. I realize that. I've just been thinking the

thermal load is really the --

VICE CHAIRMAN CROFF: Yes, and that's what really allows it. You know, I mean, at some point, getting the uranium out would then become important, because of volume considerations. But it's the heat removal that's important.

MEMBER CLARKE: If I could ask one other quick question. The RFPs that are going out for the demonstrations that will be done, they will be done at existing facilities. What are the --

MR. WYMER: No, that's a big political football.

MEMBER CLARKE: Are you talking about --

MR. WYMER: People in Idaho Falls want to build a new facility out there for the demonstration. There is already a facility built and has never been occupied at Oak Ridge National Laboratory which is called a TURF facility. It was originally designed for the uranium-233 thorium fuel cycle, which has large hot cells and waste-handling facilities and could be used in this -- within six months they could have equipment in there and -- part of it, and running.

But there is a strong political push to put the whole thing -- build a whole new facility out

in Idaho. So that issue is an issue.

MEMBER CLARKE: Okay. I understand.

VICE CHAIRMAN CROFF: Can I clarify that? I'm not sure -- are you talking about a demonstration?

MR. WYMER: Yes, the demonstration.

VICE CHAIRMAN CROFF: Okay. He's talking about the smaller demonstration facility that may or may not be helped.

MR. WYMER: That may or not come to pass, would that --

VICE CHAIRMAN CROFF: That's right. The commercial facility is going to be a big, green building.

MR. WYMER: That's a long way down the road. Yes, that's a whole new deal.

MEMBER CLARKE: And one other quick one. You didn't say anything about hydrogen generation, but is that still on the table? There was to be a demonstration at Idaho at a high-temperature gas-cooled reactor hydrogen generation. Is that still in the plan, or --

MR. WYMER: Well, it's still -- it's part of the Bush administrative initiative, you know, to replace fossil fuels with hydrogen sometime in the far distant future. That's based on thermochemical cycles

rather than electrolysis.

MEMBER CLARKE: Right.

MR. WYMER: And there are several processes that have been considered that require temperatures that you can only reach in high-temperature gas-cooled reactors of the graphite type, because you've got to get up to 800 or 900 degrees Centigrade in order to break water into hydrogen and oxygen using chemical intermediaries as sort of catalysts.

MEMBER CLARKE: I guess I just wondered if that demonstration is still on the table.

MR. WYMER: That -- it has not reached the demonstration stage yet. It is -- they are still looking at a variety of processes, and Argonne has put together a sort of protocol that -- a yardstick that they use to measure these two or three competing thermochemical cycles with respect to feasibility first, and then economics, and then -- well, there can be industrialized -- this sort of thing.

So any process that will eventually be demonstrated has to pass through this screen that Argonne has fabricated. It's a very regimented and stylized procedure that you put these processes through that measure thermodynamic efficiency and heat

loss and the whole -- everything you do in an engineering study of such a thing. So it's far, far from a demonstration at the present time, Jim.

MEMBER CLARKE: Okay. Thank you, Ray.

MEMBER WEINER: I'd like to start with a couple of observations. The first, I was very interested in your description of the graphite spheres, the pebble bed spheres. The full graphite sphere that has the little ones embedded is about the size of a tennis ball, and the PBMR in South Africa, which I'm surprised you didn't mention, circulates them and then drops them out when they're done.

The other observation is that the transuranic waste is, of course, currently stored in the waste isolation pilot plant, and the limit on that is a policy. It's constrained only by policy. The Act says it has to be defense-generated, but there is no technical limit. They could always excavate more.

MR. WYMER: I think that's right, yes.

MEMBER WEINER: Now, the questions I have is -- oh, finally, another one, it's my understanding that to get the complete factor of 10 reduction you really need the generation four reactors that burn up the actinides.

MR. WYMER: That's exactly right.

MEMBER WEINER: Yes, I just wanted to clarify that. Is the program using the information that has been gained? In some of these areas you have -- we have Fort St. Vrain, we have the PBMR, we have EBR 1 and EBR 2, and the FFTF. And all of these address one or another facet of this. Is that information being used?

MR. WYMER: Yes, it is. It is being incorporated very well I think, and I just heard the other day that the FFTF, which has been sentenced to death three or four times, is -- has been reincarnated and --

MEMBER WEINER: I thought they had started to drain the sodium.

MR. WYMER: Well, there is some left in the bottom they haven't sucked out yet. So it may yet be reborn.

MEMBER WEINER: With EBR 2, there is -- was a process to recover all of the uranium. Is that being looked at at all, that you can recover the uranium, put the rest of the actinides in a pellet, although you can segregate those, and the fission products go into salt, is that being utilized at all?

MR. WYMER: Well, as you have indicated, it was -- there was a reprocessing demonstration done

on the EBR 2 fuel out at Idaho Falls, which was a very successful operation. That is the only large-scale demonstration of this molten salt reprocessing that has really ever been done, and it was successful, in fact.

MEMBER WEINER: So that is being incorporated into the --

MR. WYMER: Yes, indeed.

MR. WYMER: -- because that was managed, as you probably know, by what we call Argonne East at the time. And the people at Argonne East, namely Jim Layler and company, are sort of leading the charge on this whole GNEP initiative and recycle initiative. So you would expect that their technology would be incorporated into the thinking, and it has been.

MEMBER WEINER: I've forgotten now who discussed the doses, the reduction in dose. Was that --

MR. FLACK: That's me.

MEMBER WEINER: When you look at doses from mining, do you count the fact that now uranium is being mined by in situ leach mining, and there's virtually no dose at all to the workers?

MR. FLACK: Well, I assumed it wouldn't have been that way back when this study was done --

MEMBER WEINER: No, but --

MR. FLACK: -- back in the '70s. So that may change the numbers.

MEMBER WEINER: But if the study is updated --

MR. FLACK: Right, I would think that that would have to reflect that fact.

MR. WYMER: Well, the problem -- if I may, the problem with that, Helen, is that there -- the way to do in situ leaching is very limited. You have to have very specific conditions. For example, you have to have a hard rock pan under the deposit, so that the acid or base you put in it doesn't go to China. You know, it's -- wind up in the groundwater that people have to drink.

So the fraction of the uranium which is recoverable by in situ leaching, while it's significant, is a minor part of the uranium ore recovery issue.

MEMBER WEINER: It's our understanding that having this -- gone to the National Mining Association meeting -- is Latif here? I guess -- yes. That most of the uranium mining in the United States at the present time is in situ leach mining, isn't it?

MR. WYMER: I would not have thought so.

Is that the case, Latif?

MEMBER WEINER: But it's -- I mean, it's a --

CHAIRMAN RYAN: It would more important -- the plans forward are for in situ leach, which is a surprise but true. Even though the recoveries are perhaps lower than hard rock mining, it's so much easier and so waste-desirable that they are going that route.

MR. WYMER: Yes.

CHAIRMAN RYAN: And all those that have expressed interest have talked about in situ.

MR. WYMER: I knew that that was the plan, but I did not know that was the chief way these days.

MEMBER WEINER: In fact, they talk about going back to hard rock mining as a sort of last resort for uranium.

Finally, I don't quite understand what you meant, Ray, by truly -- there is no truly stable inert form of iodine. Are you thinking that the iodides dissolve, which they --

MR. WYMER: Iodides dissolve.

MEMBER WEINER: Okay. So you don't consider that stability.

MR. WYMER: No, it's certainly --

MEMBER WEINER: I mean, it's a stable chemical compound.

MR. WYMER: Yes, I would like something like borosilicate glass, you know, that doesn't --

MEMBER WEINER: Oh, okay.

MR. WYMER: -- that doesn't go anywhere when you hit it with water.

MEMBER WEINER: Okay. How do you trap krypton?

MR. WYMER: The krypton can be done a couple of ways. One is you just trap it as a gas, and you compress it.

MEMBER WEINER: Okay.

MR. WYMER: And another is that -- that has been proposed and has been demonstrated on a small scale is you can ionize it and shoot it as a plasma into a surface of a metal where it's incorporated actually beneath the surface of the metal, and it's firmly fixed. So that's another approach.

MEMBER WEINER: That's very interesting. Finally, I have one more, how are we going to compete with the Russians if they are ahead of us?

MR. WYMER: We've got more money.

(Laughter.)

MEMBER WEINER: Okay.

VICE CHAIRMAN CROFF: Ruth, I wasn't sure whether Ray's response on the krypton got to your full question. Were you asking how it was recovered or the wasteform for it?

MEMBER WEINER: No. I was asking, if it's a wasteform, how do you actually trap it? And he responded to that.

VICE CHAIRMAN CROFF: Okay. Mike?

CHAIRMAN RYAN: I'm a believer on iodine. There's no such thing as solid iodine. It goes wherever it wants to go.

We had a briefing some months ago on the overall process, particularly from the waste generation point of view. Let's see, it was done by Andy Griffith from DOE. And I struggle with one of his charts where he showed uranium oxide waste as Class C waste, and iodine --

MR. WYMER: I've also seen that --

CHAIRMAN RYAN: -- iodine waste is high-level waste, tritium waste is high-level waste.

MR. WYMER: Yes, it's --

CHAIRMAN RYAN: -- and I -- it led me to this question. The devil is in the details on what is separated from what at each one of the maybe 2,000 boxes that we're going to end up with.

(Laughter.)

MR. WYMER: It may have looked that way, Mike.

CHAIRMAN RYAN: In all seriousness, that's where the waste generation is going to be determined. None of these processes are perfect. And uranium that contains something that could make it Class C could also make it true or could also make it high-level waste based on how much of the devil is in that particular detail.

So I struggle with the fact that this is not going to be as clean from a waste management standpoint as we might like to think. It could be better in some regards; it could be more troublesome in some regards.

MR. WYMER: Yes, I think you're absolutely right.

CHAIRMAN RYAN: That's a caution I throw out here.

The second caution I throw out is -- and I don't know the answer to this question -- but I would be curious to know how much plutonium in the form of MOX fuel elements we're going to produce, and whether or not we have enough reactors in the world to burn this MOX fuel, because if we don't have a ready

way to burn it, there's going to be an inventory of plutonium.

It's just going to be in a slightly different form, and that -- you know, I'm wondering if we're really solving a strategic or a safeguards issue. Unless you really understand the flow rate of -- and I don't know how much plutonium goes into a MOX fuel element and how many MOX fuel elements can you burn in a conventional reactor per year, and so forth. That flow rate has not been clarified to anybody.

MR. WYMER: Well, the rule of thumb I'm familiar with -- this may be out of date -- is that up to one-third of a lightwater reactor can be fueled with MOX fuel. And Allen probably knows more about this than anybody else in the room.

VICE CHAIRMAN CROFF: I think it's reactor-specific. Some reactors can't handle much at all because of control rod issues and this kind of thing. But let me back up to a higher level question that bears on this.

CHAIRMAN RYAN: Well, I won't ask that one, then. I'll leave that one.

(Laughter.)

VICE CHAIRMAN CROFF: When I remembered last, DOE was not planning to recycle plutonium or the

actinide product in LWRs.

MR. WYMER: That's right.

VICE CHAIRMAN CROFF: They were going to hold it in anticipation of the advanced burner reactor.

CHAIRMAN RYAN: Now, that's my next question.

VICE CHAIRMAN CROFF: So what you can do in a LWR doesn't make any difference.

CHAIRMAN RYAN: The LMFPR in the United States perhaps failed for more political reasons than technical ones. But Phoenix and Super-Phoenix are not operated. And as far as I know, fast reactors and burner reactors, which is a fast reactor by a different name, don't exist.

MR. WYMER: Russia has a couple.

CHAIRMAN RYAN: And they're working well, or not so well?

MR. WYMER: Last I knew, the BN-600 was working, but I don't try to keep up with it.

CHAIRMAN RYAN: So I wonder why the burner reactor concept isn't more prevalent at this point. Again, I'm asking questions that I don't know the answers to, but --

MR. WYMER: Why isn't it discussed more in

-- because it's -- mainly because it's farther down the road, and I think the NRC licensing problem that will hit them first by a substantial time margin will be lightwater reactor fuel reprocessing using one of these advanced processing methods.

CHAIRMAN RYAN: But the burner reactor also had some inherent material science questions and, you know, we end up with metallic sodium is the best kind of coolant and heat transfer medium, and that has its own headaches. And the neutronics are not exactly the same. I mean, the delay fractions are shorter, and control circuitry has to be tighter, and, you know, there's lots of interesting and challenging problems, but I wonder, you know, if all of that is worked out or if there has been advancement in those areas.

MR. WYMER: It is not worked out, and part of what DOE is trying to come up with now is in the short term a reactor that they can use to take small amounts, however much they can get out of these mixed actinides, and determine their burnup characteristics in a fast flux spectrum. They're casting about, and several people have sort of offered up reactors to do this.

The Canadians have offered up a reactor

they think can be used for this. The French are saying, "We'll stoke up one of our fast reactors and do it." Of course, the FFTF is now, as I mentioned earlier, rearing its head. So there is -- there are neutronics --

CHAIRMAN RYAN: We're on the leading edge of a research effort rather than a -- we're ready to build on that.

MR. WYMER: That's right. The neutronics are still to be determined, yes.

CHAIRMAN RYAN: Yes. I guess the last kind of global point I'd offer is any country that is reprocessed in this magnitude -- and I take the number of -- you know, we're building one bigger than any -- than the --

MR. WYMER: Like always.

CHAIRMAN RYAN: Every one of those countries has a much more complex waste management regulatory structure. That is, they have intermediate level waste, high level waste, and low level waste.

MR. WYMER: They do have intermediate level waste categories that they --

CHAIRMAN RYAN: With different disposal schemes, and so forth, than we're talking about. And I wonder -- you know, I -- as I sit and think about

it, I can envision ways where our current scheme could work, but it's going to take a much more flexible and interpretive approach to how you deal with high and low level waste and the waste classification system, or you could say, "Well, we really do need to become more formal and create something in the middle." I don't know that -- again, I don't know the right answer. I'm just offering this up to --

MR. WYMER: I suspect --

CHAIRMAN RYAN: -- see if these are issues we should explore in the white paper.

MR. WYMER: I think maybe you should. I think -- well, I don't know about the white paper, but I think that it's going to be an iterative process. As DOE gets farther along in their development of work and their studies, both in the burnup reactors and in developing processes, determining what the separations -- how good the separations are of these various things, which is what we're waiting for the answers on on these runs.

This will -- as these answers come out, this will provide input, I think, for the NRC to sort of continually reassess and refine what they are proposing, what their regulations are. There probably should be some latitude built into their regulations

that allows them to accommodate as yet undetermined information.

CHAIRMAN RYAN: A couple of final points. One is there's a number of these kind of economic studies from the Boston group and others that have looked at this system and have kind of given it a thumbs up as making some economic sense. And, again, with all of these other questions, not only the technical issue swings, but there are swings in the finances of all this.

MR. WYMER: You bet.

CHAIRMAN RYAN: So I would just maybe cast one little at least curious eye on some of those projections. And the final is is that, you know, a lot of the writeups on GNEP and on these kinds of approaches have GANTT charts where starting and end dates are shown as exact dates and months over a 40-year period.

MR. WYMER: We always do that. We always do that.

(Laugher.)

CHAIRMAN RYAN: But --

MR. WYMER: We were supposed to be done at 4:00, you know.

(Laughter.)

CHAIRMAN RYAN: Okay. Well, and again, so all of these points I'm raising you would consider to be at least food for thought for exploration in the white paper.

MR. WYMER: Yes.

CHAIRMAN RYAN: Again, I'm not trying to answer them today, but I think they are -- they are valid points to maybe pursue.

MR. WYMER: Yes, I think the issue of how far into decommissioning does the NRC get, and how far into plant design for proliferation resistance and this sort of thing do they get? This is a touchy issue that you'll get some -- some kickback from industry on.

CHAIRMAN RYAN: And that's fine. But, I mean, the time to maybe wrestle with some of these issues and explore them a little more fully is now rather than later when we get something up and running and we're not sure how to fix it.

MR. WYMER: You're right.

CHAIRMAN RYAN: And I'm sorry to tell you how -- that most of the Barnwell facility has been pretty much chopped up and sold as scrap, except for the one large concrete structure, which is also internally pretty beat up. But it stands as the last

testament to the effort in Barnwell.

(Laughter.)

MR. LARSON: My office is gone.

CHAIRMAN RYAN: No, actually, it's -- well, there's one of them in there, the one in the plaid.

(Laughter.)

MR. LARSON: Just a question. I thought we weren't really supposed to address safeguards in any detail as -- not only in this Committee, but in this paper. I think we talk about it, you know, in a few pages of the --

CHAIRMAN RYAN: And that's fine. I was just trying to get an understanding of the flow rate, because when you start -- you know, I mean, MOX fuel -- as you well know, in South Carolina, came in and went to Duke Power, and that was kind of an issue in the fuel element just traveling along up to one of the Duke powerplants where they're in the core now, I understand, some test elements I think.

So I just wonder, as we consider all of that, how that would --

MR. WYMER: I think it's --

CHAIRMAN RYAN: -- as storage or --

MR. WYMER: I should have said "safety"

rather than "safeguards," in the design of the plant Howard. How much of that do you build in.

CHAIRMAN RYAN: Right. And my question, really, is one of just material flow. How much plutonium are you going to burn per year in reactors that use MOX fuel, versus how much do you have in inventory or material that you're going to make into MOX fuel, and, you know, where are those materials stored, and, you know, how does that flow -- the flow through that system work?

So thanks for the discussion. I appreciate it.

MEMBER HINZE: Well, I'll try to ask a couple of pertinent questions here, and that's not easy. I'll focus on the suggested issues for ACNW consideration. I'd like to ask a very generic question. What are we going to receive in the white paper?

Are we going to have options presented to the Committee related to these various issues, and then, we will work from those to lead to what is finally in the white paper? How is that going -- what are we going -- what more kind of detail are we going to see about each of these issues coming out of the white paper specialist?

MR. WYMER: There will be some discussion of them, Bill, and depending on the particular issue you will get more or less useful information.

MR. FLACK: I think what the real purpose of the white paper is is to kind of flesh out what the issues are. I don't think the paper should explore too much as to what -- you know, leading to one more or the other. I think it's more or less to try to identify what's there and the basis of why it's there.

But I guess, is that --

MEMBER HINZE: Yes. So the Committee will not be suggesting courses of action regarding any of these. But it will just look at the range of --

MR. WYMER: Unless there's something that really just jumps out at us, Bill, that says --

MEMBER HINZE: Okay.

MR. WYMER: -- you really ought to consider, you know --

MEMBER HINZE: I'm just trying to get a feel for how much more information we're going to be getting on this. Let me move on, because you've already taken up too much of my time.

(Laughter.)

One of the items that I don't see here is the process of selecting a site. And it seems to me

that is --

MR. WYMER: The process of what again? I'm sorry.

MEMBER HINZE: The process of selecting a site. And I didn't say "site characterization" yet, because there might be such an action as, for example, volunteer sites that will come along the pike. And that would be the most opportune of the various options you can think about. And one might think about the incentives for that.

And then, there's site characterization. I mean, if I think of -- if I think of West Valley, and -- oh my gosh, if I think of West Valley and site characterization, or Morris, you know, I think that we have learned an outstanding amount about the regulations regarding site characterization as a result of our efforts with Yucca Mountain. And I would like to see site characterization as well as the process of a site specification as fairly heavy items here.

I also wonder as I look at this is, what kind of handling facilities -- those of us that think Yucca Mountain are currently in the process of thinking a great deal about handling facilities and the whole pre-closure situation. That, I think, is a

-- something -- we're going to see different forms here. You know, do we want to put the borosilicate in a TAD? Do we have to put it in the same TAD, or can we just put it out there in a virgin way?

There are certain problems there that I think would be extremely important for this Committee to identify and try to look at.

One of the things that bothers me very much about West Valley is this co-location of storage sites with the reprocessing. This, of course, has led to all kinds of problems, as we all know, at West Valley. And I think that there should be some thought given to this -- how much co-location.

When I see a storage of a 10-year period, a 10-year supply on a site, I guess if I were on the City Council of West Lafayette, Indiana, I wouldn't really encourage us to volunteer a site. What I'm saying is that there should be some thoughts as to really how much storage of waste that there is going to be on the site.

And I was thinking about this low-level waste, as all of you were talking, and then you brought it up, Ray. And I think that that -- you know, that may be the 800-pound gorilla in this whole situation. And it's not only the fact that we have to

have a place to put it, but, you know, do we really want to ship this, as we heard yesterday, 2,000 or 3,000 miles? This is going to have an impact -- the location of low-level waste sites.

So this whole business of co-location, storage on site, proximity to low-level waste facilities, the site characterization, you know, these are some of the thoughts that pop in my mind. And I have now used my 10 minutes. Is that right?

VICE CHAIRMAN CROFF: No, you've got a few minutes left.

MEMBER HINZE: Well, I think that's enough. Thank you.

CHAIRMAN RYAN: Can I ask one dumb question, Allen?

VICE CHAIRMAN CROFF: Sure.

CHAIRMAN RYAN: And it's -- again, I ask it out of ignorance. You know, I'm reminded when AEC was broken into then ERDA and NRC, and let's call it DOE and NRC to make it simple, and the NRC really had the commercial world and DOE had the non-commercial world, the military side of things.

I guess I'm trying to understand a little bit about how new efforts in reprocessing are not in the commercial sector, that they are viewed to -- I

mean, that DOE has a major role. And that's not to say they're not capable and competent and have lots of research facilities. But how -- has that been worked out? Is that an issue we need to think about? I mean, are all the laws in place that govern roles and responsibilities for the major agencies? And that was one of the regulatory slots.

What are the -- you know, the Environmental Protection Agency certainly has a generally applicable radiation protection standard obligation. DOE certainly has skills capabilities and research facilities that are significant and substantive. And the NRC has a clearly-defined role in the commercial side of nuclear energy. It's not just producing electricity and power reactors. But how is --

MR. WYMER: That's an interesting issue, all right, because DOE, when they're doing demonstrations, is self-regulating, as you know, and --

CHAIRMAN RYAN: Right.

MR. WYMER: And, still, if they do eventually build a demonstration plant, which would be the wise way to go, that's for commercial fuel. And it is not just strictly for DOE interest and

application, so they're in a gray area there. And just to what extent is that an NRC issue, because it is a demonstration plant for a commercial reprocessing plant, although it's a development plant. It depends somewhat on the scale of it, as much as anything else, how big is it.

CHAIRMAN RYAN: And how the information would flow from one to the other, if it is commercialized, and, you know, it would get, then, regulated under the list that John had, that one page. I mean, the flow of all that is certainly not clear to me, and I just think that's an area to think about.

MR. WYMER: Yes, it's kind of a gray area, really.

MEMBER HINZE: You know, there's a related area, too, in terms of like repository -- or a reprocessing plant versus a nuclear reactor -- is Appendix A on the seismic hazards. Is that still applicable in terms of the piping problems, the frequencies, and all of these kinds of things? Somebody has to take a look at that before there's a general application. And who -- and as Mike says, who is the leader of the pack in developing the standard regulations for --

MEMBER WEINER: There's an associated

problem, too, which is the pollution control from a -- from such a facility. I mean, is this, then, an EPA-regulated function, or a DOE-regulated function?

MR. WYMER: So far a demonstration facility would be DOE.

MEMBER WEINER: But, again, it falls into the same category.

MR. WYMER: It's still the same issue, yes.

VICE CHAIRMAN CROFF: I want to try to answer two different things here. With regard to EPA, I mean, DOE has to use EPA standards.

MR. WYMER: Absolutely.

VICE CHAIRMAN CROFF: I mean --

MR. WYMER: To their sorrow.

VICE CHAIRMAN CROFF: -- EPA standards trump DOE orders I guess is the way to say it. But at what scale that comes in, I mean, you know, DOE's research and development activities don't -- you know, don't get subject to that. At some point, there's an out, and I don't know where it is.

With respect to what you were asking, Mike, my impression, based on what I've seen historically, is, you know, when you get into this gray area Ray mentioned -- and there is a gray area

and this demo plant epitomizes it -- I think the first option is for DOE and NRC to work it out between themselves as to whether NRC will license it or whether DOE will do its own thing. If DOE does the regulation, then how will NRC be involved? Like looking over their shoulder to learn kind of stuff.

You know, if that can't be worked out, or if somebody else gets interested, then Congress can weigh in on it. And my example is there that Congress did weigh in in the -- I think it was the Energy Policy Act where they said that the NRC would license that demonstration reactor. I think it was the demonstration --

MR. FLACK: Well, they asked for its licensing strategy to be developed for the demonstration -- you're talking about the one in Idaho for the next generation --

VICE CHAIRMAN CROFF: Yes, right.

MR. FLACK: -- the work --

VICE CHAIRMAN CROFF: Where Congress included some language there.

MR. FLACK: That's right.

VICE CHAIRMAN CROFF: The last discussion I heard on the fuel cycle demonstration plant is that DOE would do it under its orders, but NRC would be

very involved with them. And DOE -- I think we heard that from Laidler and Buzz Savage, as a matter of fact, this last summer. But that's the way it -- but there is gray -- ambiguity there, I guess, that has to be worked out case by case.

CHAIRMAN RYAN: Fair enough. And, again, I'm not saying that we should come up with some answer or some grand plan, but it certainly is something to highlight if there are substantive issues that we can put our finger on to say, you know, how is this going to happen?

VICE CHAIRMAN CROFF: Okay. I'll take a couple of things. First, I'll extend what Mike said just a little bit. And this is on the waste classification issue. I think even given using a UREX-type process with these various different waste streams, the sort of fractionation of what we used to know as high-level waste into four or five different things, I think our existing waste classification system would really be severely strained.

In particular, and first, as you pointed out in deciding which of these things is high-level waste, you know, right now we're sort of handling this under this exemption, the real waste determination process, but -- and maybe that could be used as a

rubric to do it. But --

CHAIRMAN RYAN: If I may just on that point, Allen, it's a very good point, and if you recall, we've had many discussions on the fact that the current definitions are origin-based and they're not risk-based.

VICE CHAIRMAN CROFF: Right.

CHAIRMAN RYAN: And if there is an opportunity to start focusing on individual radionuclides, their form and their content and their individual radiological characteristics, whether it's per human exposure or environmental pathways, and so forth, this might be the opportunity to get away from origin-based definitions and go to risk-based thinking. So I just offer that as a thought.

VICE CHAIRMAN CROFF: It might be able to use the existing system, but it would take some real artwork, I think, to do it.

CHAIRMAN RYAN: Like I said, I think, you know, you could creatively do it with some of the caveats that exist now. But it would become much more of a patchwork than it already is.

VICE CHAIRMAN CROFF: And as Ray has pointed out, when you start going to pyroprocessing where there is just absolutely no equivalent to this

first cycle raffinate, I mean, the whole thing just falls part.

CHAIRMAN RYAN: And the idea of going to risk as the measure of -- you know, risk-informed measure as the way to guide regulatory development is certainly current with the way people think about things today.

VICE CHAIRMAN CROFF: Yes. John, could you take me to 48, please? I first want to make sure I understand this. What I think you said is that if we had to use the existing regulatory framework today to license the scope that Larry and Ray have talked about, the UREX+1a, that this is the regulations that would apply to the various parts of that operation. Is that --

MR. FLACK: Pretty much. I mean, it's something that right now is in place, that you would have to try to make accommodate.

VICE CHAIRMAN CROFF: Right. I mean, my first reaction is that -- I mean, that's at least ugly, if not impossible, to try to use all of those regulations on basically one integrated operation. I mean, and some of it's, you know, risk-informed, some of it's not risk-informed, some PRA, some ISA. That doesn't seem to be possible. I'm more or less talking

to the Committee here, but --

CHAIRMAN RYAN: Well, the other point that -- when this slide came up that I thought about is, okay, this is what regulates the facility perhaps, and let's assume that's right and true. What regulates the waste that goes out the door? What if you create a waste you can't get rid of? So 61 and 63 are on the table again.

And, you know, we heard earlier, you know, in talking about things this week that, you know, if you create a waste that you don't have an outlet for you're in trouble. And that could happen. And by the way, this doesn't even raise the dimension of chemical waste or mixed waste. That's a whole new add-on to, you know, your list. So I would just maybe make a note to add those three.

MR. LARSON: Well, and Ray mentioned training. You know, Part 55 applies. If it's a Part 50 license, then the operator has got to be licensed under Part 55. And in the paper we discuss, you know, the failure rates, which were pretty high. You know, like 60 percent over a five-year period of those that were licensed or attempted to license by the NRC failed.

CHAIRMAN RYAN: The operators.

MR. LARSON: Right. The operators.

VICE CHAIRMAN CROFF: And one important point. I mean, we know that DOE is proceeding at some pace with an EIS on greater than Class C. And they've got some current vision of what falls in the greater than Class C category, and it's sort of some oddball and relatively small volume stuff. If this GNEP thing proceeds, that's going to change that equation radically.

What we call "greater than Class C" or call it "true waste," it's, you know, the same thing, but there's going to be a lot more of it and it's going to be a very different waste. And it seems to me that that issue, and what these transuranic wastes might look like, need to be on their screen, so they can consider it in the EIS.

CHAIRMAN RYAN: Let's add an additional view there, Allen. If you look at the origin-based definitions, that's based on processing technology that came out or experiences of the processing technology that came out of Hanford and Savannah River mainly I guess.

So the origin-based definitions are really chemical engineering process efficiency-based definitions. How much can we really get at? When

does the first pass solve an extraction -- you know, that has most of the stuff that you're interested in in it become second pass, and not so important in its waste?

So greater than Class C in the context of what we're talking about now doesn't mean much in terms of risk yet. So what's actually in it? Is it high risk, is it low risk? It's not -- I mean, to me, greater than Class C is just a convenient metric. It's got -- it's not necessarily directly related to risk.

VICE CHAIRMAN CROFF: Oh, no, I didn't mean to imply that. I --

CHAIRMAN RYAN: And I think that's an additional dimension we have to kind of remind ourselves of to think more about.

MR. WYMER: I think you don't want to understate the importance of the tension that's going to exist between the regulators and the builders and operators of these plants. You know, originally, there was not a strong incentive to have a very high -- a really high recovery of plutonium. It was purely an economical decision. What's the value of plutonium? And is it okay if you leave one percent of it behind where it shouldn't be, you know, in a waste

where -- where you really would rather not have it?

And as far as industry is concerned, those are financial decisions. They're not regulatory decisions. And there's going to be a lot of give and take here, it seems to me. You've got to protect the public, but you've got to allow industry to proceed.

VICE CHAIRMAN CROFF: I think the way this is going to have to play out is, I mean, if you look at that flow sheet, in a regular world a lot of that is not economic. I mean, like separating cesium and strontium --

MR. WYMER: Yes.

VICE CHAIRMAN CROFF: -- and this kind of thing. And the owner of the spent fuel is going to be DOE, and DOE is going to have to write an RFP that has the specifications on what is recovered. And that's what the industry will bid on, or not as they choose.

MR. WYMER: Or not, yes.

VICE CHAIRMAN CROFF: Let me go on into another thing. On the EPA standards, I think there -- you know, EPA started a job in 40 CFR 191. They didn't really finish that job, and that raises an issue: is the EPA going to continue to look at, in particular, carbon-14 and tritium limits or not? They said they were going to, and they did not. Are they

going to revisit krypton and iodine? That was done many years ago and done using the microdose to mega people approach is exactly what they did. And no bones about it. That's what was in the analysis.

So there is a need to understand what the EPA is going to do or not, and then the -- for the NRC to figure out what it's going to do. One thing I stumbled across just yesterday is compliance with 40 CFR 190 is explicitly mentioned in 10 CFR 20. So it's on the books. I mean, it's integrated already. It just says, you know, you will do it. I mean, there's no further elaboration.

Can -- I don't know -- Ray or Larry tell me, what's the difference between UREX+1a and GANEX? I mean, when you stand back and look at them, they seem to end up producing about the same product streams.

MR. WYMER: I'll take a shot at it.

VICE CHAIRMAN CROFF: Except for the cesium and strontium.

MR. WYMER: Yes, well, the technetium also is not taken out as a separate stream. Aside from that, it's pretty much the same thing. The French have just simplified the process. They have not put as many process steps in it. They're not as ambitious

as -- with respect to putting a whole lot of separation steps one after the other as we are. They're much more -- I think much more practical and pragmatic in how they're proceeding.

VICE CHAIRMAN CROFF: Yes. Okay. Any followup questions from the Committee?

MEMBER WEINER: I have one. It's kind of coming back to something that Allen has said. There are processes of chemical safety with all of these processes, particularly with the waste processes. And I think this, again, poses a regulatory concern. Is this going to be under OSHA? Because presently I believe most DOE facilities are not, they are self-regulated.

MR. WYMER: Most of these reagents are not highly toxic reagents. They are toxic, sure, but they're not -- they're not in the extremely toxic category. You'll have to be careful, and they'll have to be -- if you do incinerate them, which would be one way to dispose of them, then you'll have to go through -- all the whole ritual that the toxic incinerator went through down at Oak Ridge where they were very carefully regulated, they sampled the off-gas to make sure they weren't producing carcinogens, and so there will be a whole series of things to be done in

handling these organic materials.

MEMBER WEINER: It's not so much the toxicity of the reagents, but the hazards associated with the chemical reaction and on that kind of a production scale.

MR. WYMER: You're talking about safety.

MEMBER WEINER: Yes, it's a chemical safety.

MR. WYMER: Yes, most of these things are not -- do not have a lot of latent energy in them. They are not highly explosive things. I can think of one exception, and it's not in any of these flow sheets, and that's an ion exchange separation that was practiced at Savannah River using separation of plutonium as a plutonium nitrate complex. Perfectly safe. As long as you kept it wet, they left it on an ion exchange column until it dried out and it exploded.

So there are things you have to be careful about when you have, as I would call it, latent energy involved. There are not many of these processes that posses that kind of potential chemical reactivity.

MEMBER WEINER: I guess the reason I raised the question is not so much for the demonstration project. I'm sure that would be very

well controlled. It's if you start to do this on the production scale, then you -- then you start to get lax and start to have -- just the risk associated with doing anything on a production scale.

MR. WYMER: To use the sort of expression, that's when you start plowing up the snakes.

MEMBER WEINER: Yes.

MR. WYMER: When you actually get in there and run the process. And you've got to be willing to have a development staff to deal with those poisonous snakes that you're plowing up.

MEMBER WEINER: The other comment, very briefly, that I'd like to make is the -- what is required in an environmental impact statement has certainly developed since 1976. And there is a lot more -- a lot more detail and a much more prescribed format required now.

MR. FLACK: That's true, and I think part of that whole effort is to look at alternatives as well. I mean, that's part of -- you know, which one is going to give you the best. Is it worth pursuing? But I think, in general, NRC adopts the environmental impact statement when it's satisfied with it.

I know we brought up the issue about doing GESMO, NRC getting back involved in that, and the

attorneys commented on it, saying that, "Well, it's really a DOE thing. We may get engaged in it, as it's developed, but it's not ours. It should be a DOE initiative."

VICE CHAIRMAN CROFF: In the summer meeting, Buzz Savage acknowledged that DOE had the ball on a generic environmental impact statement. Now, I have no idea whether anything is going on, but they agreed they had the ball, so --

CHAIRMAN RYAN: I just took a quick look, and the process hazards analysis standard would apply, because it applies to any place that has 500 pounds of nitric acid. So we're in the game.

(Laughter.)

MR. TAVLARIDES: Excuse me, if I may, but that to me was something that I was thinking about is the nitric acid solutions that you have. And if you do any concentrating of that, then you may end up getting dinitrates and possibilities for explosion.

VICE CHAIRMAN CROFF: And you do concentrate nitric acid recovery.

MR. TAVLARIDES: Yes, exactly.

VICE CHAIRMAN CROFF: Bill, you had a question?

MEMBER HINZE: Very quickly. In terms of

the reprocessing process, does it require a great deal of water? Is this something that one should be concerned about?

MR. FLACK: I don't know. Ray, is that --

MR. WYMER: Yes, there's a lot of water.

MR. TAVLARIDES: Yes, there is water in -- there are washing streams and

VICE CHAIRMAN CROFF: Well, let's be clear on the question. I think you were asking whether there's a continuous water consumption, and I think it's relatively small. Once they get it in the plant, most of it is recycled.

MEMBER HINZE: Okay. So it's not very --

VICE CHAIRMAN CROFF: With that, are there any questions from staff?

DR. HAMDAN: A quick one, if I can.

VICE CHAIRMAN CROFF: Okay. I'll give you a quick one.

DR. HAMDAN: When you mentioned the significance increase in the waste volume, and I'm not clear, are we talking about -- you mentioned ten -- perhaps ten-fold increase, and how we talk about Barnwells, Yucca Mountains, and what timeframe are we talking about?

MR. WYMER: Your question relates to

timeframe?

DR. HAMDAN: Yes. I mean, number one, is it a low-level waste site, or -- Latif Hamdan, NRC staff. So, really, this increase in volume that is expected, a) what timeframe are we talking about, and b) how many waste sites do we need? And are they Yucca Mountains, or are they Barnwell?

MR. WYMER: Will somebody rephrase that question for me?

VICE CHAIRMAN CROFF: Let me try and actually answer it. The amount of waste going to a Yucca Mountain will, if all this happens as projected, would decline. That's why they're doing all the fractionation.

MR. WYMER: Yes, by about a ten-fold.

VICE CHAIRMAN CROFF: By about, you know, 10x. Now, what would increase is you've got to manage some cesium and strontium. You're going to have transuranic waste that will require disposal, and a number of other things.

MR. WYMER: Yes, you'll have kind of a plethora or a wealth of smaller waste streams than we have at the present time.

DR. HAMDAN: So you are talking about TRU waste that is going to increase? Transuranic?

MR. WYMER: No, there will not -- there will not be a net increase in waste. It will be about the same. You know --

VICE CHAIRMAN CROFF: I mean, you're comparing spent fuel assemblies to something that's fractionated in a lot of little streams. So I'm not sure we can compare it right now.

MR. WYMER: Yes, we're not destroying mass anywhere here, except in a little fissioning that is going on. So there will be about the same mass of waste there ever was, but it will be parceled out differently.

VICE CHAIRMAN CROFF: Mass of radionuclides.

MR. WYMER: Yes.

VICE CHAIRMAN CROFF: The transuranic waste and a lot of --

MR. WYMER: Yes, radionuclides.

VICE CHAIRMAN CROFF: -- and that kind of stuff.

DR. HAMDAN: Thank you.

DR. ABU-EID: Good afternoon. My name is Bobby Eid. Just a comment on the question. Just to remind the Committee that the international community, IAEA, they are developing new guidance on waste

classification, and currently it is being reviewed by the staff, just for your information. And there is --

VICE CHAIRMAN CROFF: Excuse me. We became aware of that I think yesterday or the day before. And we're going to ask for a briefing on whomever of your staff goes over. We're very interested in it.

DR. ABU-EID: Okay. That's one thing, just to remind you in this regard. What issue is dealing with is actually a norm classification, whether to include the norm or not. That's one issue that we are dealing with now, but there are other issues, too. But the good news is that risk is being used, just to let you know.

The other thing is, just to remind you, that certain countries, like Japan for example, they do consider the spent fuel as a resource rather than a waste. That's the reason there is what's called the Joint Convention, and the Joint Convention is on the safety of spent fuel management and the safety of waste disposal.

That's the reason, because there are differences about the classification of the spent fuel, if it is waste or a resource, and that's one of the reasons actually they have the Joint Convention.

Just for your information, to take it into consideration.

My question and recommendation regarding about regulatory framework, and the framework is regarding 10 CFR Part 50, Appendix I, and if you want release limits, that most likely it was mentioned that we could apply the current regulations and guidelines for NRC for the processing of spent fuel.

So in this regard, if this is the case, just to remind you that the current guidance using ICRP-2 for dose conversion factors, or for the dose factors for that, and there is inconsistency with 10 CFR Part 20. And I would add this as a recommendation or an issue to be considered, such that if we had the consistency or if there would be more update of the regulations, to consider this kind of inconsistency with 10 CFR Part 20.

CHAIRMAN RYAN: Bobby, I'd second that thought and remind everybody that for long-lived persistent radionuclides, like plutonium and the other actinides and some fission products, that the difference in doses calculated from ICRP-2 versus the current committed dose approaches are exacerbated. They can be up to a factor of 50 times different, and the longer lived material is, in fact, forgiven more

than the short-lived material.

You know, we calculated annual doses from plutonium in the old scheme, so 5 rem per year translates to a committed dose in the new scheme of 250 rem. So it's a very significant numerical question which has implications. But under our current scheme of using committed doses for internal exposures, everything is the same every year.

You start out each year with a clean slate, in other words, and that frankly is, in my own personal view, the appropriate way to do it. So there are some significant changes when you begin to see actinides and other long-lived species that persist in the body for decades or more, in terms of this question that Bobby is pointing out again. So --

DR. ABU-EID: Thank you.

CHAIRMAN RYAN: -- it's not a trivial matter at all.

DR. ABU-EID: Thank you.

VICE CHAIRMAN CROFF: I think at this point I'd like to just take a couple minutes and describe how I see this going forward, so that it answers a question Ruth asked the other day. And it was a good question, but I wanted to defer it until now.

What we're promised at this point is in early December NRC will -- staff will come in, will send us a -- their paper on how they would propose to regulate fuel recycle, which of these things they think is the best way to go out of some of these options we outlined.

I hope we get that before our next meeting. And assuming it's out, we're promised a briefing on that in the next meeting. And I would like to see if I can get Ray up here for that, if possible, and, of course, John will -- he will be here anyway, I hope. And so that will take us into December.

At that point, we're going to try to -- and we're going to be working on the white paper in the interim, and leaving a couple of blanks. At that point, I'd like to get a good, clean draft of it, and in early January send the white paper out for -- I'll call it stakeholder review.

In other words, to the Committee, but also to people like NMSS and other interested parties, to get their review of it, get the comments back in, and make some revisions in it before our February meeting, make the final revisions in it so the Committee has got a clean white paper. And I will be, at that time,

trying to draft some kind of a letter for consideration in our February meeting.

Beyond that, I think our letter, looking at what we discussed today, even -- is going to be far from definitive with answers on all of this. Some things will have some recommendations, others we're going to have to bore into. And I'm looking at this issues list as sort of a framework for additional briefings or working group meetings into the future. We'll figure out what the highest priority topics are and get people in to help educate us on whatever.

So that -- yes, but at that point, to finalize the white paper and get that done and not let that continue to drag on, because, you know, every meeting you get more information that can go on forever. So that's my present plan.

CHAIRMAN RYAN: Just a couple of clarifying points there, if I may, Allen. I think, you know, I'm reminded that one fool can ask more questions than a thousand wise men can answer. So our white paper -- I think, you know, we need to identify issues where we think things are clear, and I think the second part is we need to focus on issues and at least identifying issues where we think things are not so clear.

You know, for example, the question that Bobby and I just discussed is very straightforward on how you fix it. The question of, do you fix it or not, is the only uncertain part. But what needs to be done is crystal clear.

There are other areas like, you know, the ones we talked about in terms of, you know, how the various agencies are going to share the obligations at the top level. That's clearly not clear, and perhaps above our paygrade. That's something outside of our, you know, area of charter and responsibility. But identifying it I think is appropriate.

So we're really in the business of identifying areas where we think things are clear, and, you know -- and, again, all in the framework of the basic context that our team has laid out today. And let me add my thanks to all three of you for doing a great job of giving us a four-inch firehose to learn as much as we can about reprocessing in a couple hours.

But is that, you know, making sense?

VICE CHAIRMAN CROFF: Yes.

CHAIRMAN RYAN: Okay. Great. And I think when we talk about, you know, NMSS and others it would be helpful, as we ask them for input, to be a little

bit more explicit about what we're looking for and the context in which we're looking. You know, we're not asking them to give us answers. We're asking them to say, "Do we have all the questions they think are important in our white paper? And have we at least put the framework for the question out on the table in a smart and accurate way?"

VICE CHAIRMAN CROFF: Yes. Okay, thanks. With that, I'm done.

CHAIRMAN RYAN: Okay.

VICE CHAIRMAN CROFF: I'll turn it back to you.

CHAIRMAN RYAN: Once again, thanks to Ray and John. We really appreciate -- and Howard, of course, yes. We really appreciate your efforts in putting together the history. Well, he has been so quiet. He has just kind of been taking notes. We welcome you back, sir, but I appreciate all your efforts, and we'll look forward to moving this to the next step.

Thank you all very much.

With that, why don't we take a 10-minute break, and then the Committee will reconvene.

I think our remaining activities are letter-writing, so I believe we can conclude the

record here today. Is that correct? So we'll conclude the formal record here today, and we'll take up a couple of letter items when we reconvene at 4:20.

(Whereupon, at 4:11 p.m., the proceedings in the foregoing matter went off the record.)

CERTIFICATE

This is to certify that the attached proceedings before the United States Nuclear Regulatory Commission in the matter of:

Name of Proceeding: Advisory Committee on Nuclear Waste
174th Meeting

Docket Number: n/a

Location: Rockville, MD

were held as herein appears, and that this is the original transcript thereof for the file of the United States Nuclear Regulatory Commission taken by me and, thereafter reduced to typewriting by me or under the direction of the court reporting company, and that the transcript is a true and accurate record of the foregoing proceedings.

Charles Morrison
Official Reporter
Neal R. Gross & Co., Inc.

Dose-effect relationships and estimation of the carcinogenic effects of low doses of ionizing radiation

Medical Avisor
Nuclear Plant Operation
Electricité de France

President of Health and Research section
French Radiation Protection Society

French academic report

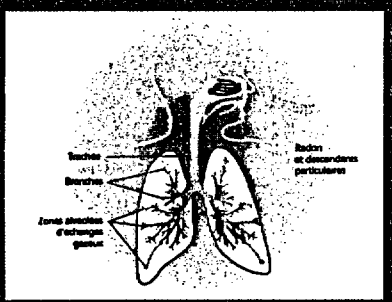
– Over the past 20 years the French Ministry of research has twice asked the Academie des Sciences to carry out a critical review of the available data regarding the effects of low doses of ionizing radiation on health.

– In 2003 the two Academies decided to join their *effort* for an update of two main topics:
  - the dose–carcinogenic effect relationship
  - the carcinogenic effect of low doses.

– A working party was set up; its report was accepted after a few modifications suggested by the reviewers and it was released in March 2005.

Comparison

- The main problem for both medical and non-medical uses of ionising radiation is the possible carcinogenic risks associated with small doses of ionising radiation.

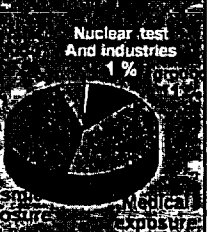


- These eventual risks are also of great importance with regard to natural irradiation,
  - for example it would be of great value to assess the risk of lung cancers caused by various radon concentrations in the air at home or at work,
  - and whether there is a practical threshold below which the risks become negligible.

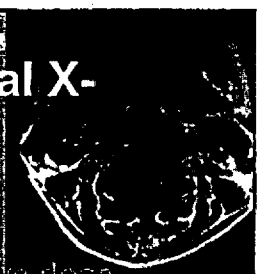
Assessment of carcinogenic risks

- The assessment of carcinogenic risks associated with doses of ionizing radiation from 0.2 Sv to 5 Sv is based on numerous epidemiological data.
- however
  - the doses which are delivered
    - during medical X-ray examinations
    - or the dose received by nuclear workers
    - or in regions of high natural background irradiation

  are much lower(from 0.1 mSv to 20 mSv).



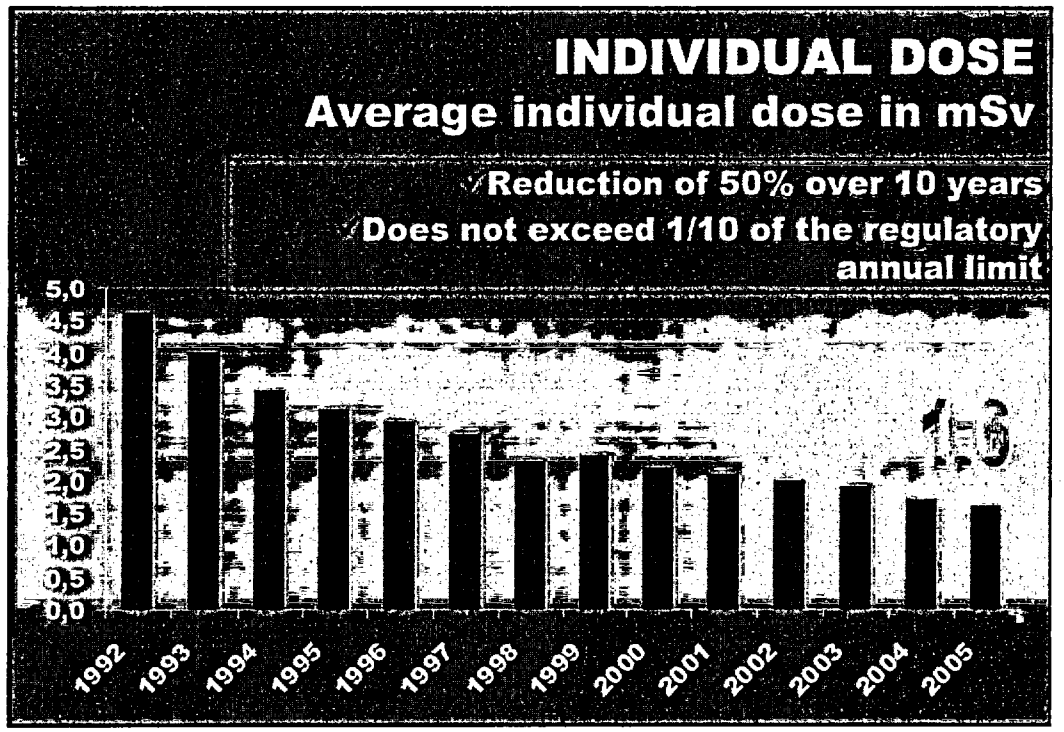
Doses delivered during medical X-ray examinations

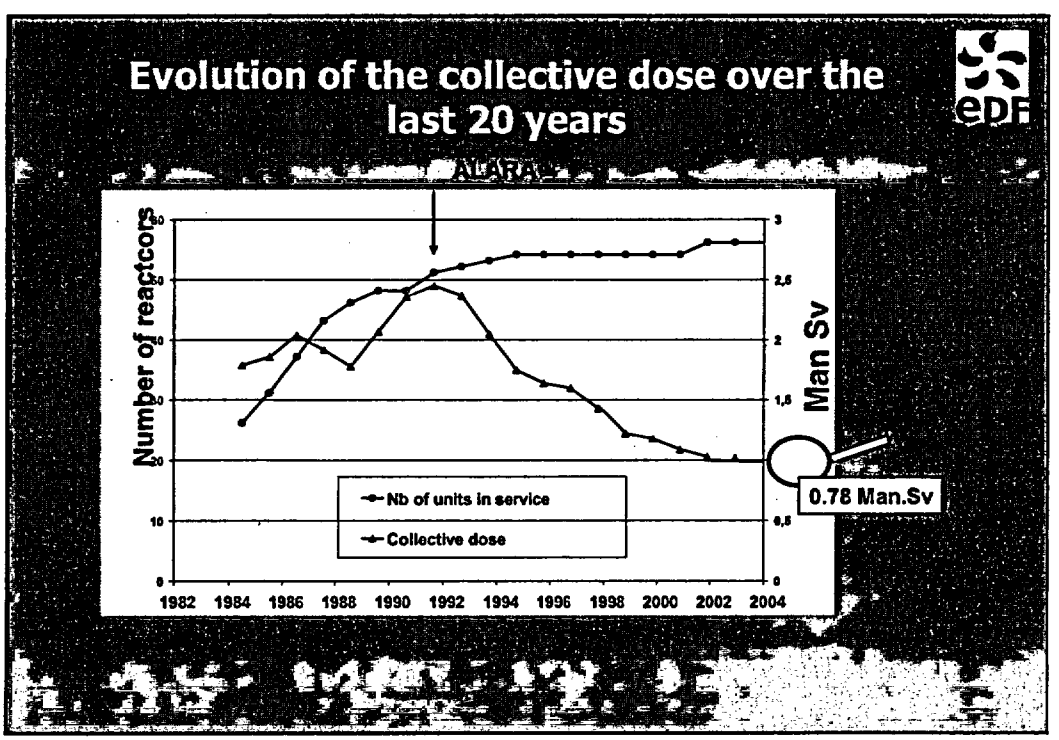


Chest X Ray	0,2 - 0,5	0,015 - 0,15
Rachis X ray	4 - 28	1,5
Urography IV	40 - 60	3
Body scan	30 - 60	4 - 10
mammography	7 - 25	0,5 - 1

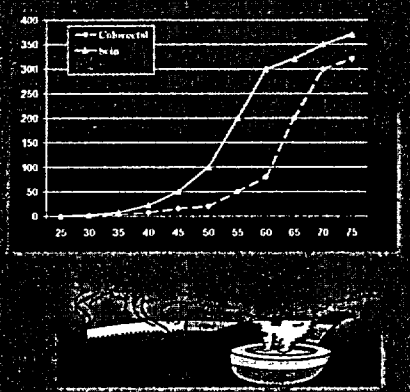
Exposure due to a chest X ray variability in Europe:

Country	skin dose mGy
Netherland	0,13
Italy	0,14
United Kingdom	0,19
Belgium	0,38
France	0,47
Greece	1,93


INDIVIDUAL DOSE
Average individual dose in mSv
Reduction of 50% over 10 years
Does not exceed 1/10 of the regulatory annual limit
5,0
4,5
4,0
3,5
3,0
2,5
2,0
1,5
1,0
0,5
0,0
1992
1993
1994
1995
1996
1997
1998
1999
2000
2001
2002
2003
2004
2005


Evolution of the collective dose over the last 20 years
edf
ALARA
Number of reactors
Man Sv
Nb of units in service
Collective dose
0.78 Man.Sv
1982
1984
1986
1988
1990
1992
1994
1996
1998
2000
2002
2004

Difficulties to detect excess of cancer



- **Following small doses, no excess of cancers has been detected; however, the lack of an increase does not exclude the possibility of a small excess of cancers.**
  - Solid tumours and leukemia have a spontaneous incidence that is high and varies according to lifestyle.
  - the possible increase in this incidence following irradiation is relatively low, so the studies must have sufficient statistical power, which requires large cohorts.
  - but, in large populations confounding factors are present and they must be taken into account by appropriate statistical methods, because their specific effect can be much greater than the effect of radiation.

difficulties with épidémiological studies

1000 mSv	500 people
100 mSv	50.000 people
10 mSv	5.000.000 people

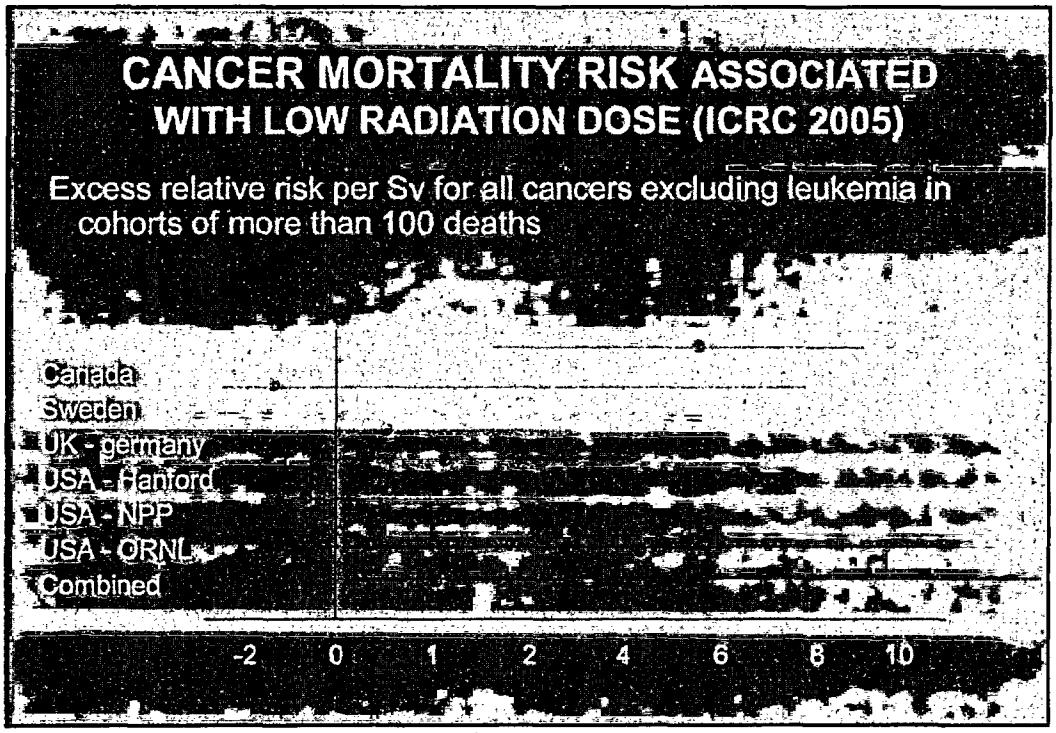
• at sea level	0,25 mSv / year
• Mexico (2240 m)	0,80 mSv / year
• medium	0,9 mSv / year
• Espirito Santo (Brasil)	35 mSv / year
• Limousin (french area)	1,20 mSv / year
• French St Alban water	1,25 mSv / year

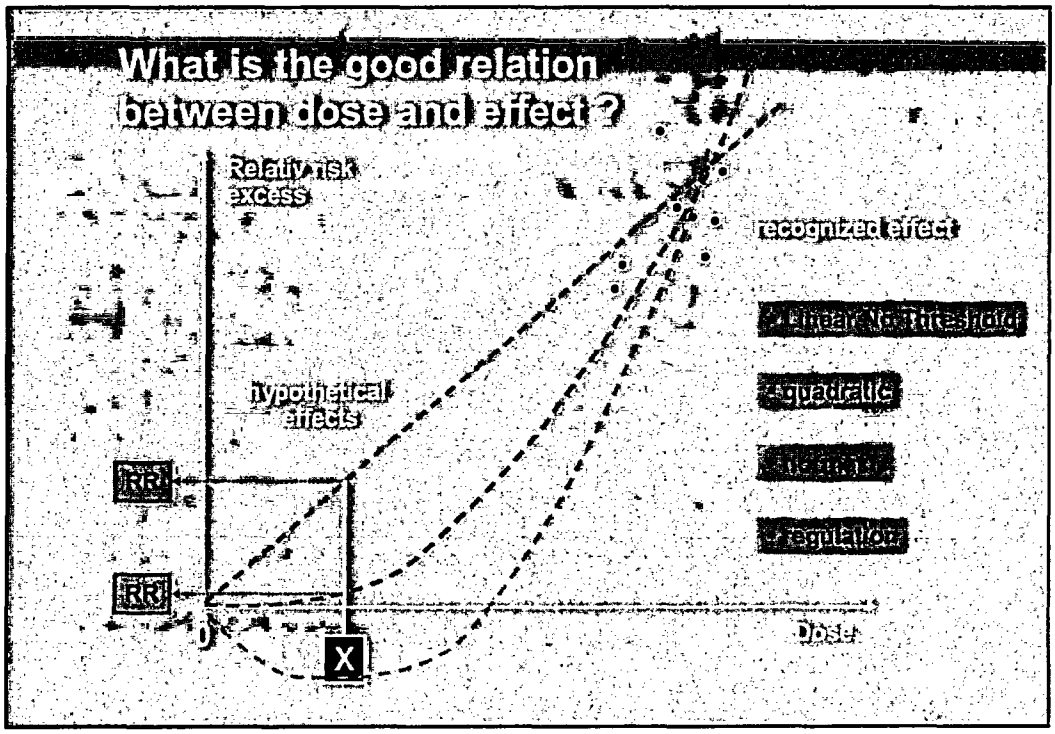
Epidemiological studies

Hiroshima-Nagasaki 76.000 ; M 200 mSv	leukaemia # 150 mSv solid cancers NS < 100 mSv
[illegible] 96.000 Nuclear Workers	leukaemia NS ≤ 400 mSv solid cancer NS
[illegible] 600.000 Nuc. W. 19.4mSv	leukaemia and solid cancers NS < 100 mSv, 1-2% K due to IR
[illegible] 220.000 ; 10 - 50 mSv / an	leukaemia NS solid cancers NS
[illegible] 47.000 ; 1,5 - 6 mSv / an	leukaemia NS solid cancer NS (melanoma)

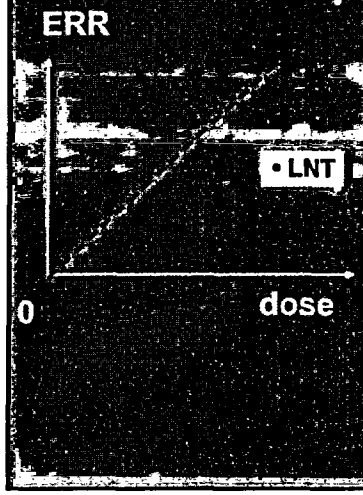
Epidemiological studies

[illegible] [illegible]	Leukaemia NS Breast cancer > 100 mSv
[illegible] 7.700 breast cancer	NS if session ≤ 150 mSv solid cancer NS
[illegible] 100.000 ; ...70 mSv / an	Leukaemia NS solid cancer NS
[illegible] 100.000 ; 2 - 6 mSv / an	Leukaemia NS solid cancer NS


CANCER MORTALITY RISK ASSOCIATED WITH LOW RADIATION DOSE (ICRC 2005)
Excess relative risk per Sv for all cancers excluding leukemia in cohorts of more than 100 deaths
Canada
Sweden
UK - germany
USA - Hanford
USA - NPP
USA - ORNL
Combined
-2
0
1
2
4
6
8
10


What is the good relation between dose and effect ?
Relativ risk excess
recognized effect
Linear No Thresh old
quadratic
regulation
hypothetical effects
RR1
RR2
0
X
Dose

A linear no-threshold relationship (LNT)



- The LNT model was used in 1956 by Russell to evaluate the radio-induced mutations in the germ cell line in the mouse.
- it was introduced between 1960 and 1980 for the purposes of regulation in radiation protection with regard to all mutagenic and carcinogenic effects in humans
  - At that time, LNT was considered a convenient pragmatic relationship but not a model based on scientific data.

In the 1960s, the International Commission on Radioprotection (ICRP) introduced it because it allows the addition of sequential irradiation delivering low or high doses of radiation received by an individual whatever the dose rate and the fractionation.

- Thus it greatly simplifies accounting in radiation protection. However, gradually LNT was interpreted as meaning that the carcinogenic risk is proportional to the dose and that **even the smallest dose induces a cancer risk.**

French academic report

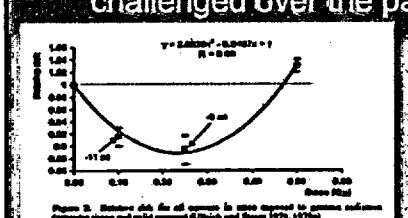
- Epidemiological studies do not have sufficient statistical power to determine risks from low dose exposures. Therefore, fundamental mechanistic studies are essential to understand biological short and long term effects of low dose IR and to help evaluating risks at those dose levels.
- Recent research developments and in particular molecular approaches have lead to new findings that put into question some of previously established radiobiological paradigms and concepts.
- The present review outlines, what we got to know recently-what we still like to know of low dose and low dose rate effects -and the possible consequences for radiation protection.

Implicit assumptions on which the use of LNT has been based for assessing the carcinogenic effect of low doses

- 1- In the range of the doses and dose rates under consideration; there is no physical, chemical or biological interaction between the effects caused by the various tracks of ionising particles in a cell.
- 2- Any absorbed dose of energy in a cell nucleus leads to a proportional probability of mutation.
- 3- The probabilities of successful repair or misrepair (per dose unit) are always the same, whatever the number of lesions in the same cell.
- 4- There should be no impact of dose or dose rate. Similarly, the probability of apoptosis does not vary with dose.
- 5- Any DNA lesion has the same probability of giving rise to a cancer, irrespective of the number of other lesions in the same cell and the neighbouring cells.

Contre a dogma

- the LNT has been used for assessing the effect of low and very low doses.
- This procedure has become a dogma in many radiation protection circles, but the validity of the LNT has been challenged over the past decade for two main reasons:-

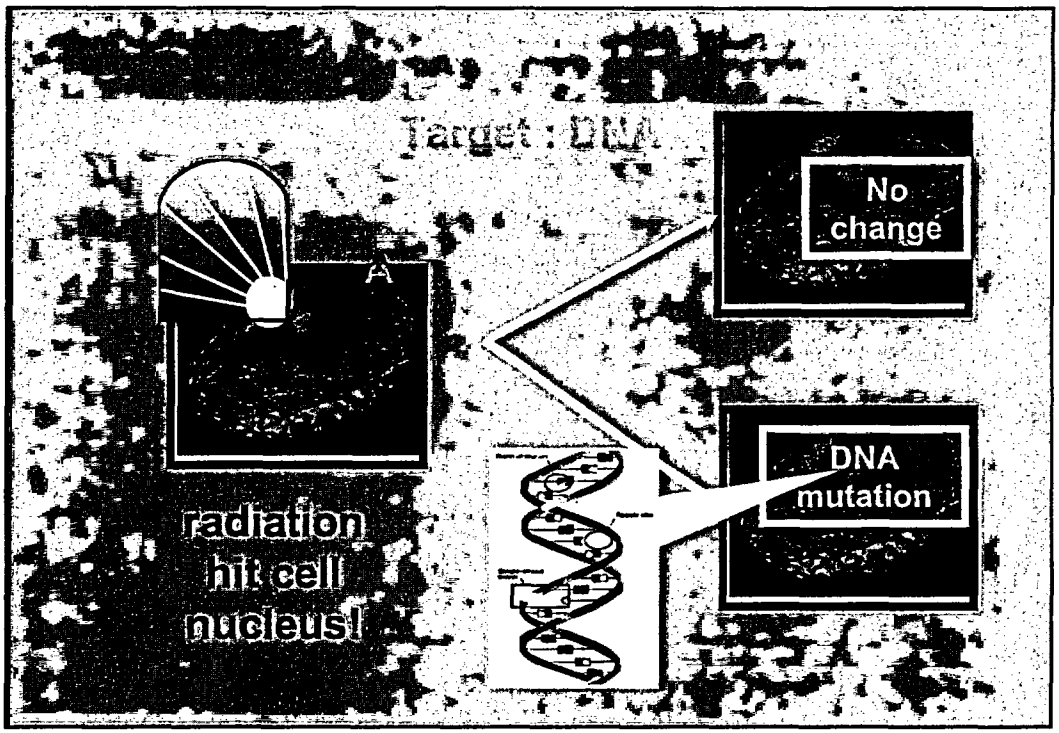


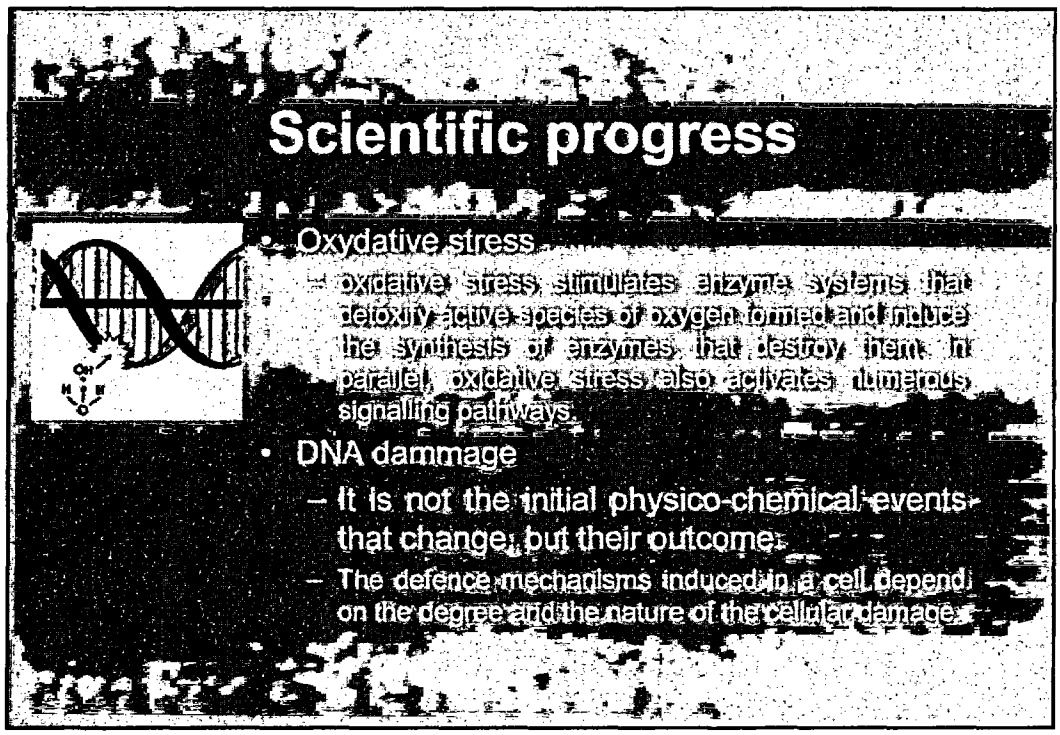
- meta-analyse on all data published
- # 60 000 mouses
- hormesis : 40 % experimental series
- doses ≤ 200-250 mSv

(a) the meta-analyses of the animal data have shown the absence of any carcinogenic effect of doses below 100 mSv,

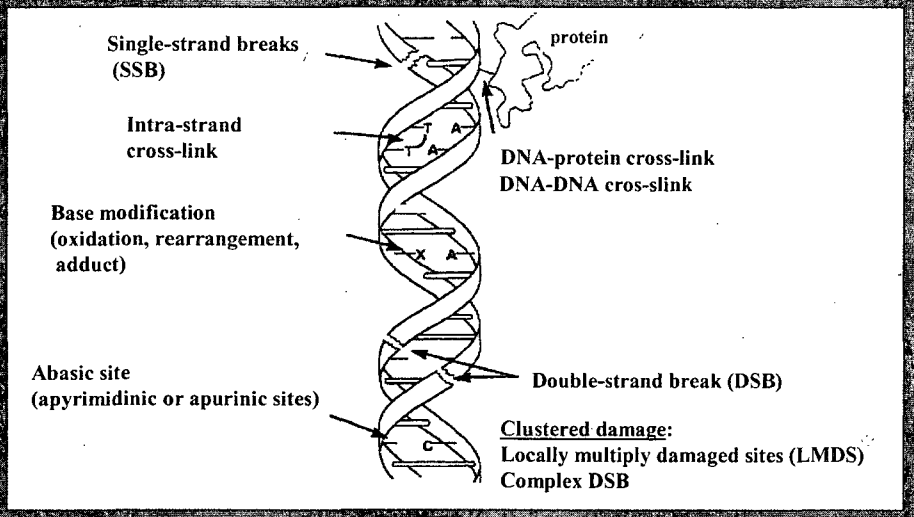
(b) scientific progress has revealed the complexity of carcinogenesis and the diversity and effectiveness of the responses of a cell to radiation.

- Indeed, a cell is not passively affected by the accumulation of lesions induced by ionising radiation. It reacts through several mechanisms.


Target : DNA
No change
DNA mutation
radiation hit cell nucleus!


Scientific progress
• Oxydative stress
– oxidative stress stimulates enzyme systems that detoxify active species of oxygen formed and induce the synthesis of enzymes that destroy them. In parallel, oxidative stress also activates numerous signalling pathways.
• DNA dammage
– It is not the initial physico-chemical events that change, but their outcome.
– The defence mechanisms induced in a cell depend on the degree and the nature of the cellular damage.

DNA damage



Differences between endogenously and IR-induced DNA lesions

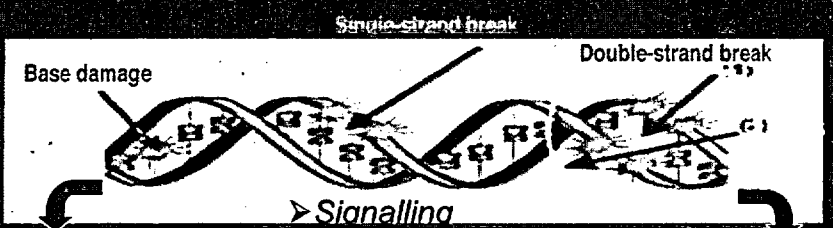
- Endogenously, due to cellular metabolism, one finds many SSBs and modified bases, however, also DSBs or complex lesions.
- IR-induced lesions in DNA include considerable amounts of DSBs and complex (clustered) lesions such as locally multiply damaged sites (LMDS), together with many SSBs and base damages.

Comparison between endogenous and radiation-induced DNA damage

DNA damage	Spontaneous lesions/cell/day	Radiation-induced lesions/Gy
Base loss	12 600	?
Base damage	3 200	2000
DNA/DNA-crosslinks	8	30
DNA-protein crosslinks	a few	150

(Burkart W et al. CR Acad Sci III 1999; 322:89-101; Ward JF Prog Nucl Acdids Res Mol Biol. 1988; 35: 95-125)

Response to ionizing radiation (IR)



DNA repair

Error-prone

Error-free

Mutation

Survival

Genetic instability

Cell transformation

Interaction of ionizing radiation (IR) with living matter

In recent years some new findings have alerted radiation biologists:

1. K-shell activation by low LET IR and the emission of two energetic Auger electrons (250 and 360 eV) can induce complex DNA damages like DNA double-strand breaks (DSBs) (Boissière et al. 2004)
2. Also, very low energy electrons (< 10 eV) can give rise to DSBs (Boudaiffa et al. 2000)
3. High LET- and low LET ionizing radiation can give rise to locally multiply damaged sites in DNA (Goodhead1994, Nikjoo et al. 2001)

Definition of clustered lesions and LMDS

•Two or more DNA lesions formed within one or two helical turns of the DNA molecule at the end of a single radiation track.

•LMDS composed of closely spaced DNA single-strand breaks (SSBs), oxidative base damage (apurinic and apyrimidinic sites), sugar and base modifications, DSB, involving opposite DNA strands.

•Predicted from biophysical model calculations (Monte Carlo) (*Goodhead D.T. IJRB 1994;65:7-1; Nikjoo H. et al. Radiat Res. 2001; 156: 577-583)*

•Thought to be induced at higher yields at high than at low LET radiation.

•May also be formed by bursts of Auger electrons (350 eV) from K-shell activation of carbon atoms *(Gobert FN et al. IJRB 2004;80:135-145)* and dissociative attachment of low energy electrons *(Huels MA et al. J.Am.Chem.Soc. 2003;125 (15): 4467-4477))*

•May constitute particular obstacles to cellular repair *(Nikjoo H. et al. Radiat Res. 2001; 156: 577-583).*

Are clustered lesions or LMDS of special interest in radiation protection?

1. In contrast to lesions arising during normal cellular metabolism, clustered lesions or LMDS are thought to constitute molecular markers or signatures of IR and to be rather exclusively induced by IR (see BEIR VII report). In addition, 30% of DSBs are of complex form (Nikjoo et al. IJRB 1997; 71(15):467-83))
2. LMDS are thought to be responsible for most genotoxic effects such as lethality, mutations, chromosome aberrations, cell transformation and cancer !! (BEIR VII).

- Much work has been done in recent years to better define and quantify these lesions in irradiated cells and to determine their biological consequences.
- See publication of Sutherland et al. PNAS 2000; 97: 103-108.; Gulston et al. NAR 2002; 30(15):3464-72; Yang et al. DNA Repair 2006;5(1):1323-34), Soucher D. et al. 2006, Radiat. Environ. Res. (in press)

LMDS and radiation risk

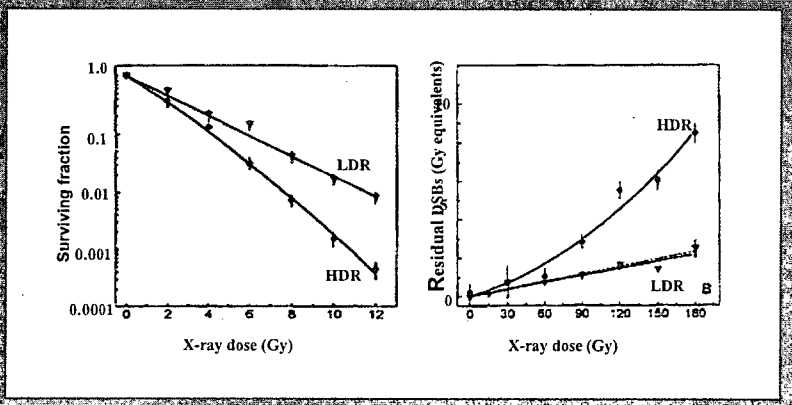
- According to BEIR VII p54: 'LMDS (clustered damage) may be viewed as complex lesions associated with IR and not with endogenous oxidative processes. If they are refractory to repair, the risk to humans posed by IR may be viewed as greater than that posed by endogenous oxidative stress.'

- However, LMDS are difficult to quantify in human cells and their number (if present), is quite limited. Most of clustered lesions may consist of complex DSBs.
- In most cases, clustered lesions are found refractory to repair but those lesions are lethal and non mutagenic.
- They are thus unlikely to contribute significantly mutagenic and carcinogenic risk of IR for humans.

DNA repair

- Differences in the efficacy of the protection system are supported by various experimental or clinical data,
- *Repair and dose rate*
- At equal doses, the mutagenic effect varies markedly with the dose rate.
- When the dose rate increases, the mutation frequency after having passed through a minimum increases strongly

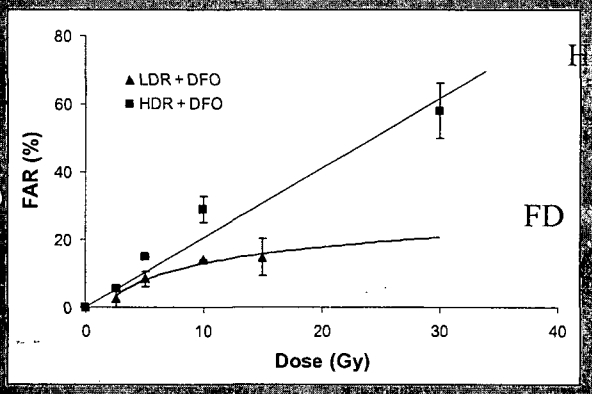
Dose-rate effects on cell survival and the induction of DSBs in mammalian cells



HDR= 4 Gy/min, LDR= 40 mGy/min

(E. Dikomey, I. Brammer, IJRB 2000; 76:773-781)

Dose-rate effects of gamma irradiation on the induction of DSB in mammalian cells (CHO-K1)



HD

FD

FAR : Fraction of activity (DNA fragments) released

Gamma irradiation:
LDR: Low dose rate (0.05 Gy/m
HDR: high dose rate (3.5 Gy/min

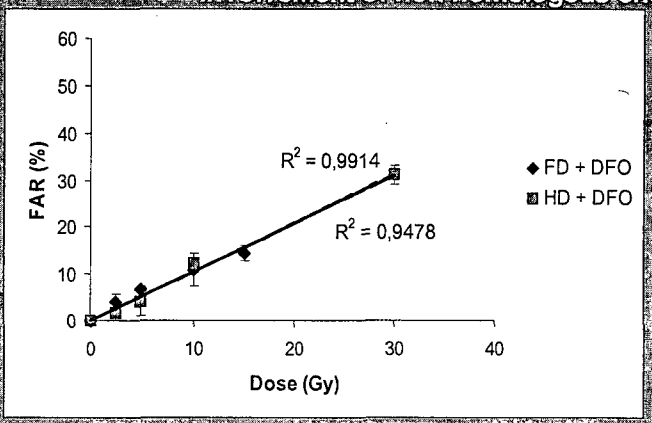
Induction of DSB is reduced after exposure at low dose-rate (LD) (0,05 Gy/min)

as compared to exposure at high dose-rate (HD) (3,5 Gy/min)

(De Boucher et al. Can. J. Physiol. Pharmacol. 2004; 82: 125-134)

Induction of DSB in a repair-deficient Chinese hamster ovary cell line (xrs6) by low and high dose-rate gamma irradiation

Involvement of non homologous end-joining



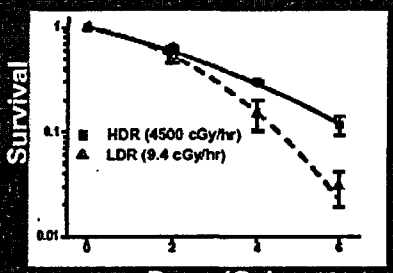
FD= 0.05 Gy/min
HD= 3.5 Gy/min

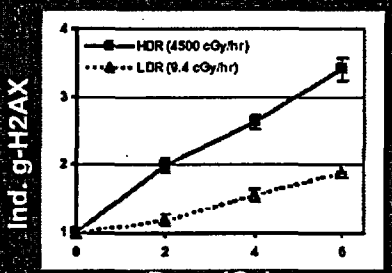
FAR : Fraction of activity (DNA fragments) released

Absence of a dose-rate effect on the induction of DSB due to the absence of repair (NHEJ) in this cell line (xrs6).

Absence of ATM activation and DNA damage signaling at very low dose rate

(Collis et al. JBC 2004; 279:49624-49632)





Taking the activation (phosphorylation by ATM) of the histone H2AX **as indicator for radiation-induced DSBs,**

Collis et al. (2004) have shown that at a very low dose-rate (94 mGy/h), DSBs are recognized by detector proteins (MRE11-RAD50-NBS1) but not repaired because of **an absence of activation of ATM**, i.e. an absence of DNA damage signaling. Signaling of DNA damage (DSB) depends on dose-rate.

---> At higher dose-rates DNA damage signaling is taking place.

There appears to be a threshold for ATM dependent signaling and DNA repair

The effect of radiation dose-rate

- DNA damage (DSBs) signaling via ATM and H2AX phosphorylation was found to be absent at a very low dose-rate (1.5 mG/min) - and associated with lethality-but present at slightly higher dose-rate (4.16 mGy/min) and at high dose-rate (750 mGy/min)

  (Collis et al. JBC 2004; 279:49624-49632)

- **Dose-rate changes affect genes of radiation-induced apoptosis** (APO-1, TRAIL, TRID etc..) but **not genes of cell proliferation** (MDM2,BTG2, ELK4, SNK, etc.)

  (Amundson et al. Mol Cancer Res. 2003,1; 445-452.).

---> Thus, exposure at very low dose levels of chronic radiation may cause more cell killing than that estimated from extrapolation at higher doses.

DNA Repair

Several well-defined pathways exist for the repair of radiation-induced lesions

High fidelity repair	*DNA damage*
Direct ligation of SSB	Single strand breaks
Repair of mismatched bases	Base mismatches
Base excision repair (BER)	Modified bases and SSB
Nucleotide excision repair (NER)	Bulky adducts
Homologous recombination (HR)	DSB, LMDS (?)
Low fidelity repair	
Non homologous endjoining (NHEJ)	DSB, LMDS (?)

Hyper-radiosensitivity

- Low dose hypersensitivity (Joiner et al. 2001, Marples et al. 2004) is observed in many cell types.
  ---> high lethality at a few hundred mGy followed by radioresistance at doses over 0.5 Gy.
- It involves poly(ADP-phosphoribosyl) transferase activity (PARP 1), ineffective cell cycle arrest in G2-phase cells and DNA repair.

- The possible role of hyper-radiosensitivity responses in radiocarcinogenesis (0-100 mGy) is not yet understood.

Low-dose hyper-radiosensitivity

- For some cell types, mortality is very high (per dose unit) at the onset of irradiation (during the first 200 mGy), then falls to a very low level before increasing again.
- This low-dose hypersensitivity is observed in many cell types leading to a high mortality rate, per dose unit, for doses of less than a few hundred mGy of low LET irradiation.

  and the mortality rate per dose unit then becomes very low before increasing again.

Variations in DNA repair efficiency

depend on the genetic background

==> individual hypersensitivity
due to mutations or polymorphisms of DNA repair genes
in the general population (OGG1, XRCC1 etc.)

==> defaults in damage **signaling and repair** are often associated with cancer predisposition:

ATM==> lymphoma, breast cancer
BRCA1/BRCA2 ==> breast and ovarian cancer
LigIV--> immune deficiency

- **depend on the differentiation status of cells and tissues.**
- **depend on age**

Individual sensitivity and polymorphisms in DNA repair genes

- Among patients undergoing radiodiagnostic (tomographic) examinations or radiotherapeutic treatments some patients have been recognized with decreased DSB repair capacity *(example: Löbrich et al. 2005: PNAS)*
- Several other studies point to the involvement of repair gene polymorphisms such as XRCC3, XRCC1 and XPD in the accumulation of genetic effects (micronuclei) in individuals chronically exposed to exposed IR.

and
were found associated with radiotherapy-related malignancies in survivors of Hodgkin disease *(Mertens et al. Cancer 2004)*

Biochemical characterization of repair pathways is important for:

- DNA damage signaling is necessary for DNA repair

- deficiencies in DNA repair are associated with cancer

- deficiencies in DNA repair are associated with individual hypersensitivity

- deficiencies in DNA repair may cause premature ageing, neurodegeneration and immunodeficiency.

Experience on yeast

- Studies carried out with the DNA micro-array technique [Mercier 2004] in yeast show that continuous irradiation, at a dose rate of 20 mGy/h, (i.e. lower than the level of irradiation that causes a detectable (lethal, mutational) biological effect), is enough
  - **to change** intracellular signaling without modifying the genome
  - **to activate** or **inhibit** numerous genes involved in the general metabolism and in defenses against ionizing radiation.

Induction of genes is dose and dose-rate dependent

At very low doses (1 mG) some genes involved in DNA repair are not yet induced. However, genes of energy metabolism and oxidative stress are induced at doses 1000 times lower than those needed for the induction of mutations (in yeast).

(Mercier et al. 2004 Nucleic Acids Res. 2004 Jan 13;32(1):e12.).

Furthermore, **some genes regulated by p53**

- (CDKN1A, GADD45A, MDM2) are induced linearly with radiation doses between 20 and 500 mGy,
- some genes involved in DNA repair are sensitive to dose-rate (XPC, DDB2), others (ERCC1 et MDM2) are insensitive.

(Amundson et al. Mol Cancer Res. 2003, 1:445-452).

Low doses of gamma irradiation (10 mGy) elicit different gene sets than high doses (2 Gy) in normal human skin cells

(Franco N. et al. Radiat. Res. 2005; 163: 623-635)

•Specific molecular responses are triggered in cultured primary keratinocytes from adult skin at low doses (10 mGy) or at high doses (2 Gy) of gamma rays.

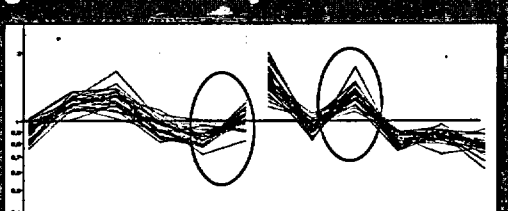
• Using DNA microarrays (10500 gene probes), it is shown that among 853 modulated probes, **the expression of 214** are specifically modulated by low dose **(10 mGy) and 370 genes** are specifically modulated by high dose **(2Gy)** exposure.

•Low dose specific genes (140 known genes) include mostly genes of homeostasis, cell communication, signaling, membrane, cytoskeleton, RNA and protein synthesis, chromatin, energy metabolism, stress, cell death and transport but **rarely DNA repair genes**.

Conclusion ==> The radiation response at low dose is rather specific and quite different from that obtained at high dose.

Clusters analysis

regulation on 3 days



Most of low dose response genes are modulated at late incubation times (48 and 72 hours), whereas most of high dose responsive genes are already modulated at relatively early incubation times.

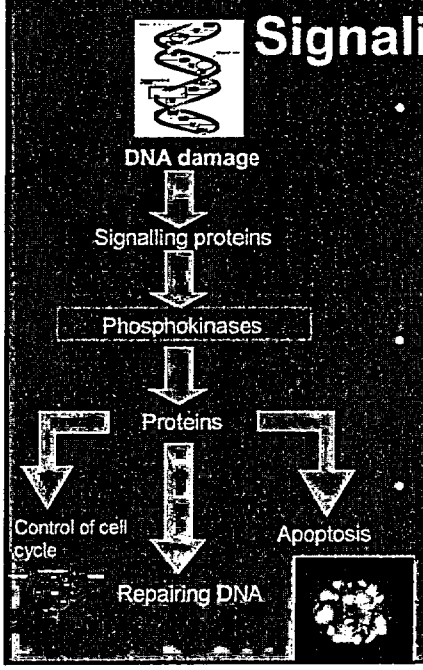
Different phosphoproteomic profiles in human fibroblasts after low- and high-dose X-irradiation

(Yang F et al. J Proteome Res. 2006;5:1252-1260)

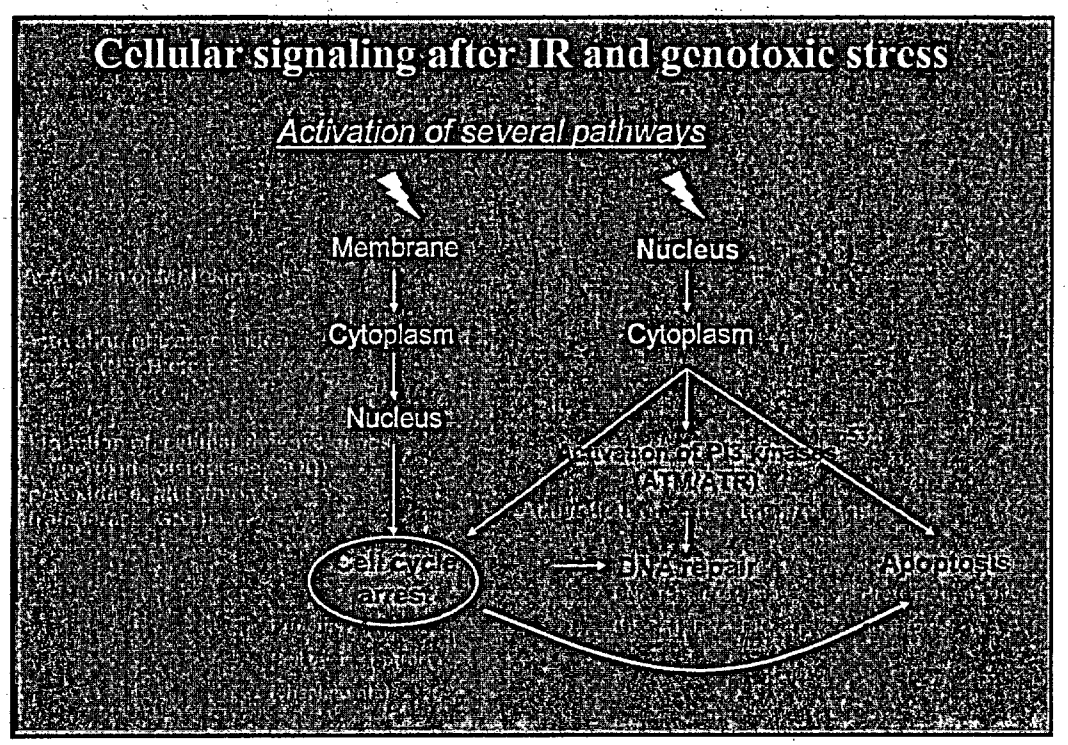
increased phosphorylation of proteins involved in cell signaling pathways and apoptosis

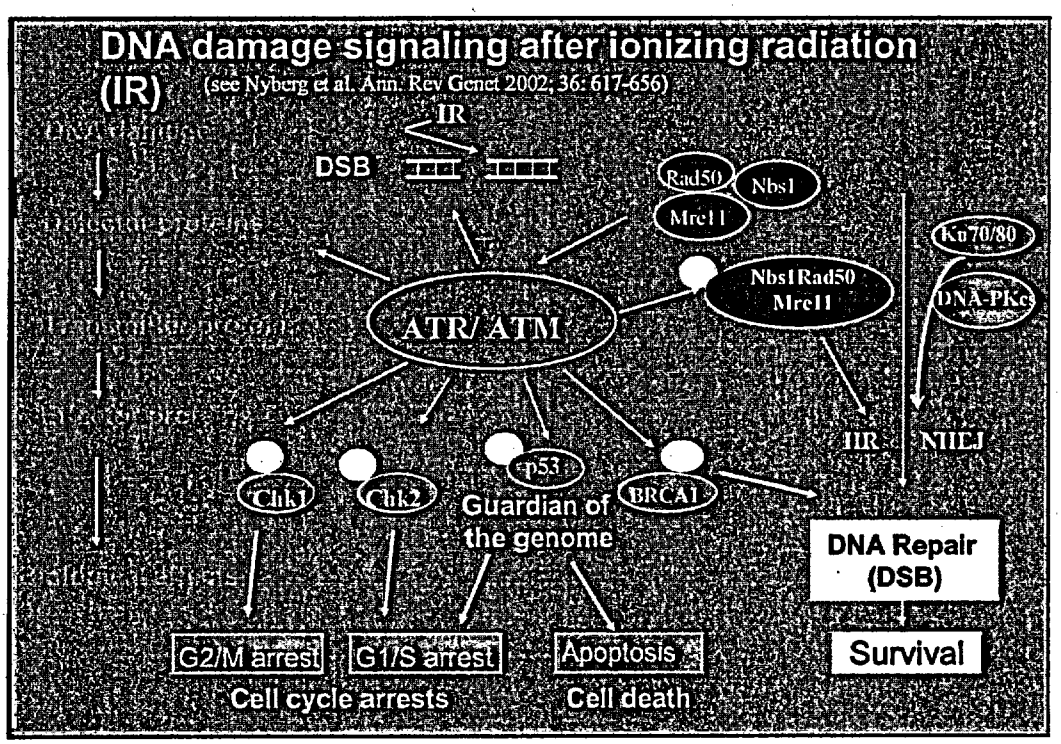
increased phosphorylation of proteins involved in more general biological processes

Signalisation in the cell

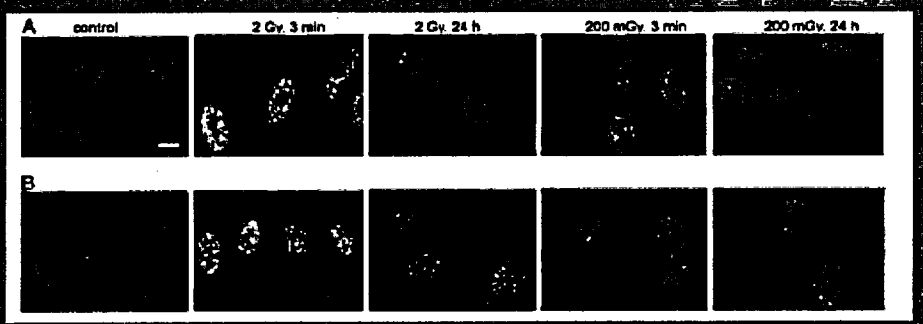


- DNA damage or modifications of the chromatin are detected by signaling proteins.
  - **The activity of these proteins is modulated by the number of lesions** (and therefore by the dose, the dose rate and LET) and by messages from neighbouring cells.
- These proteins activate phosphokinase transmitters, in particular the protein encoded by the ATM gene and the ATR gene.
- In turn, these transmitters modulate the action of proteins involved either in **cell cycle control** (the interruption of which promotes repair) and **DNA repair**, or in **triggering apoptosis**.


Cellular signaling after IR and genotoxic stress
Activation of several pathways
Membrane
Nucleus
Cytoplasm
Cytoplasm
Nucleus
Activation of PI3 kinases
(ATM/ATR)
Cell cycle arrest
DNA repair
Apoptosis

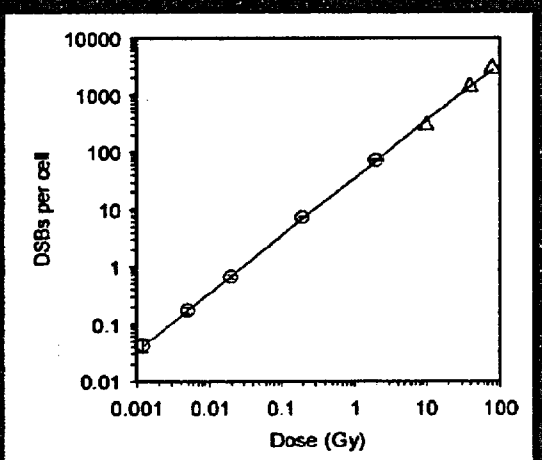

DNA damage signaling after ionizing radiation
(IR)
(see Nyberg et al. Ann. Rev Genet 2002, 36: 617-656)
IR
DSB
Rad50
Nbs1
Mre11
Ku70/80
DNA-PKcs
Nbs1Rad50
Mre11
ATR/ ATM
IIR
NHEJ
Chk1
Chk2
p53
BRCA1
Guardian of
the genome
DNA Repair
(DSB)
G2/M arrest
G1/S arrest
Apoptosis
Survival
Cell cycle arrests
Cell death

Induction and repair of DSBs as visualized by γ-H2AX



(Rothkamm K, Löbrich M, Proc Natl Acad Sci USA 2003,100:5057-5062).

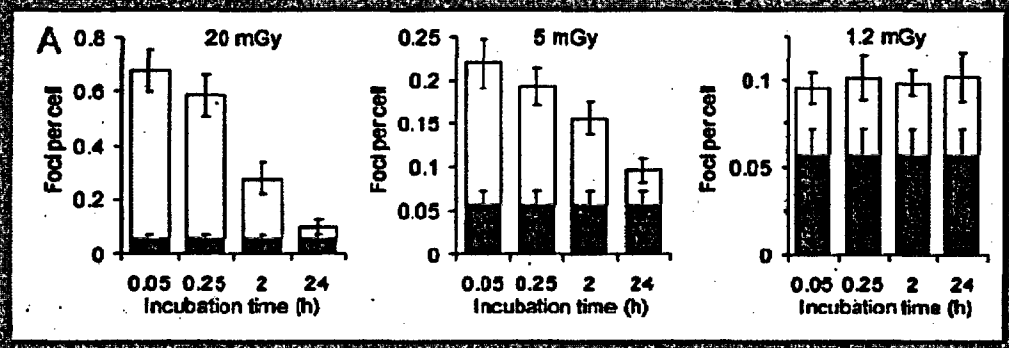
Induction of DSBs in DNA increase linearly with dose of IR



DSBs detected as γ-H2AX in human fibroblasts already at 1 mGy

(Rothkamm and Löbrich , PNAS 2003;100:5057-5062)

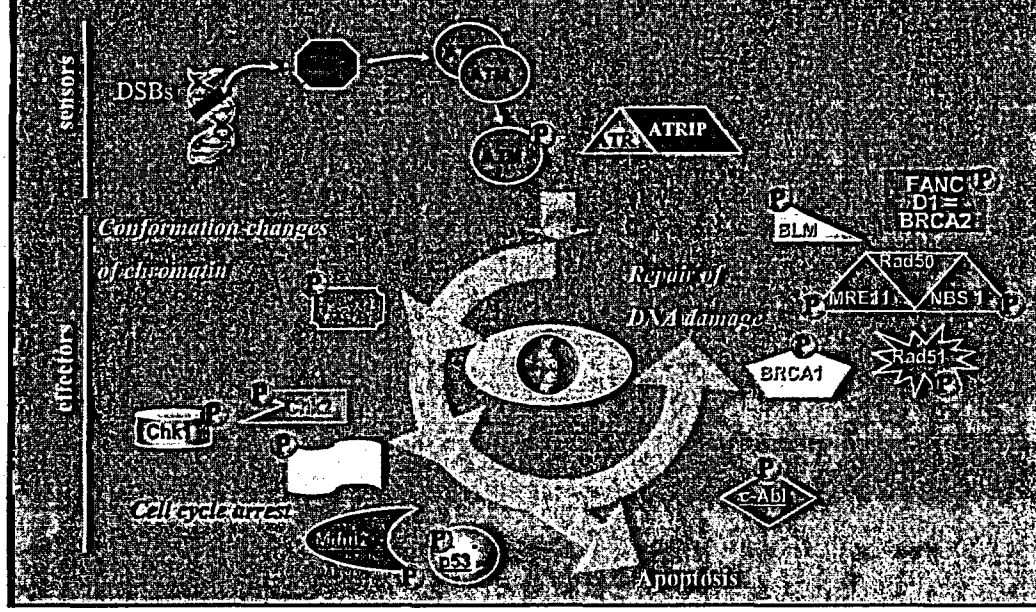
Repair of DSBs in human fibroblasts depends on IR dose and the answer is not linear with the dose



(Rothkamm and Löbrich . PNAS 2003;100:5057-5062)

Signaling of IR-induced DSBs

(see Bakkenist CJ and Kastan MB, Cell 2004;118:9-17)



Cellular reactions and DNA repair depend on the dose level of IR

(Rothkamm and Löbrich , PNAS 2003;100:5057-5062)

At very low dose (1 mGy), cells are going to die because no DNA signaling and there is no initiation of DNA repair of DSBs (or other complex lesions).

At slightly higher doses (5-20 mGy), DNA repair is initiated

(5 mGy: 1 electron track/cell —> 5-10 damaged bases,2,5-5 SSBs and 0.25 DSBs, see BEIR VII report)

At medium doses (~200 mGy), DNA repair starts to be counteracted by apoptosis.

Consequences at the tissue level

- Cells are usually imbedded in tissues.
- At very low IR doses, if a few IR damaged cells do not survive and are eliminated ⇒ tissue functions are not compromised.
- At higher doses, a substantial fraction of cells is damaged.
  - Tissue functions cannot be anymore assured except if most cellular damage is repaired,
  - and cells are allowed to survive (even if mutated) and fulfil some of their tissue functions.
  - This, however, **may also allow genomic instability, malignant transformation and cancer to occur.**

Dose-effect relationships in radiation biology are affected by non targeted and delayed effects

- Adaptive responses *(Rigaud and Moustacchi Mutat Res.1996; 435(2):127-34))*
- Bystander effects *(Mothersill and Seumour Nature 2004; 4: 256-63)*

--->Microdosimetric calculations based on target size of single cells do not correspond to the reality of radiation-induced effects

- Genomic instability *(Murnane and Sabatier, BioEssays 2004;26(11)1164-74)*
- Low dose hypersensitivity *(Chalmers et al. IJROBP, 2004;58:410-419), Marples et al. Rad. Res. 2004;161:247-55)* and
- Hyperfast early cell responses (*Fernet et al. IJRB, 20000;76:73-84; Ponnette et al. IJRB 2000; 72:1233-1243)*

Adaptive radiation response

Adaptive responses have been shown to reduce DNA damage, mutation induction, chromosomal aberrations, micronuclei and cell transformation *(Rigaud and Moustacchi, Mutation Res. 1996).*

- Priming doses of less than 5 mGy or greater than 200 mGy yield very little adaptation *(Wolff 2002).*
- Adaptive response on micronuclei production in human fibroblasts after a priming dose of 1 mGy and a 2 Gy challenging dose has been observed *(Broome et al. 2002) (needs to be confirmed).*
- Induction of adaptive responses in human lymphocytes appears to be quite variable in different individuals. Occupational exposures of 2.5 mGy/year for up to 21 years resulted in variable adaptive responses in lymphocytes challenged with 2 Gy *(Barquinero et al. 1995).*

--->The molecular mechanisms of adaptive responses are not well understood, especially, for both priming and challenging doses of 1-50 mGy.

Bystander effect

- In multi-cellular organisms, in particular vertebrates, the fate of an irradiated cell depends upon signals emitted by neighboring cells (gap junction, bystander effect, contact inhibition, proliferation control mechanisms by means of cytokines).
- Normal cells appear to be capable of inhibiting the development of potentially malignant clones. Conversely, non irradiated cells can become cancerous in the vicinity of highly irradiated cells.
- Besides an inhibitory effect (such as contact inhibition), or a stimulation of cell division, intercellular relationships can also elicit damage in neighboring cells, which have not been irradiated;

Bystander effects

Effects of radiation on single cells influence the responses of adjacent non-irradiated cells

- Often cell-to-cell contacts are required:
  bystander effects observed in human keratinocytes at gamma-ray doses of 500 mGy (Mothersill and Seymour 1997)
- In some cells bystander effects are obtained without cellular contacts (Seymour and Mothersill 2000).

The bystander effect may be beneficial or detrimental depending on the cell type and the range of doses analysed.

- JB. Little 2000 showed for very low doses of alpha particles that more mutations (of the spontaneous type) were induced in the very low dose range, whereas there were only very few deletions induced.
- After exposure to low-dose X-rays, it leads to the death of cells in which the repair of DNA damage is defective.

It is possible that bystander effects play a role below 1 mGy, where few cells are actually damaged by irradiation.

—> Are there bystander effects *in vivo* and in radiation therapy? What about abscopal radiation effects?

—> Yes, they may arise, but they need to be clearly delineated from assuming that bystander effects affect radiation-induced carcinogenesis.

Radiation-induced genomic instability

(Murnane and Sabatier 2004)

- Genetic instability is influenced by the p53 gene
  - it can be reduced by free radical scavengers
  - it is apparent at low doses and occurs at a frequency of about 3-9/1000 cells per cell/mGy after X-rays involving

Key points are **point mutations, chromosomal aberrations, telomere loss (giving rise to non reciprocal translocations)**

- It is associated with IR-induced leukemia (depending on the mouse strain) and to DNA repair defects (DNA-PKcs).
  - Excess of leukaemia in A-bomb survivors appears to correlate with excess of complex chromosome aberrations (translocations), possibly associated with telomere dysfunction (in patients with Hodgkin's disease, M'Kacher et al. 2005); telomere shortening,
  - Saturation at 10-30% at low doses (Limoli et al. 1999)

—> The influence of genomic instability on the low dose-response relationship for carcinogenesis is not yet well defined.

Non-targeted effects of ionising radiation may have positive consequences

(Belyakov OV et al. Mutat Res. 597(1-2)43-9))

- Non-targeted effects of ionising radiation might be interrelated and possibly have a protective role under *in vivo* conditions.
- These effects might relate to adaptive response because of increased non-targeted differentiation in irradiated samples.
- Based on these experimental data the authors proposed a theory that the main function of the non-targeted effects is to decrease the risk of carcinogenesis in a multicellular organism exposed to oxidative damage (including radiation induced)

Low dose radiation-induced mutagenesis

Dose-response relationships for [illegible] are not precise at very low doses (< 20 mGy).

- [illegible] are induced linearly or with a linear quadratic relationship down to 200 mGy (Thacker 1992).

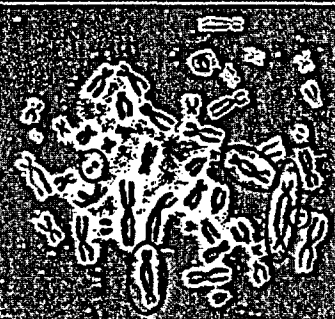
- Linear non threshold responses were observed [illegible] (except reverse mutations (pink eye unstable locus)) down to 10 mGy (Schiestl. Et al. 1994).

- Induction of chromosome aberrations (dicentrics in human lymphocytes) is linear down to a maximum of 20 mGy (Lloyd et al. 1992), for translocation down to a maximum of 50 mGy

⇒ this adds to the difficulty of extrapolating genotoxic radiation effects down to very low doses.

Chromosome aberration

LNT cannot be used to predict chromosome aberrations for very low doses. A threshold is conceivable.



Dicentriques + anneau

- The occurrence of a chromosome aberration is much increased when there are two or more DNA DSBs in the same chromosome or neighbouring chromosomes, making it possible that the rejoining of the fragments either does not restore the molecule to its initial condition

- The probability of such **error-prone endjoining** therefore depends on
  - the number of breaks simultaneously present in a limited volume,
  - and therefore [illegible]
  - and is not proportional to dose but to the square of the dose.

Radiation effects of low doses

- Below 10 mGy, the biological responses are less clear.
  - •In this very low dose range there is a much more sensitive interplay of biological processes and phenomena than at medium (200 mGy) and high doses (>1Gy).
  - •In other words, at very low doses (<10mGy) many different biological processes are activated or modulated, whereas at higher doses main stream processes like cell cycle arrest, DNA repair or apoptosis become predominant and fully determine the cellular radiation responses.

Different challenges for cells at high and low doses of IR

High doses: Genes induced concern [illegible] Cellular programs are directed to get cells survive (even at the dispense of error-prone repair) or to die (apoptosis, mitotic death..)
Responses are directed by relatively few parameters such as:

- •number of cells hit in the tissue,
- •activation of genes involved in DNA damage
- •signaling and repair and/or initiation of cell death pathways (due to excess of damage).

Low doses: Genes induced concern general metabolism and broad spectrum responses. Many factors and parameters can interfere with the regulatory network of the overall response. The responses are verysensitively linked to cellular reactivity:

- •sensoring and detection of changes in structure and function of important cellular constituants,
- •metabolic states (redox and energetic status),
- •state of differentiation,
- • cell cycle progression, cellular communication etc.

- For risk evaluations the qualitative and quantitative influences of these cellular factors and parameters have to be defined: Genetic and physiological predisposition of cells and tissues, state of differentiation... etc.

A new concept in radiation biology emerged....

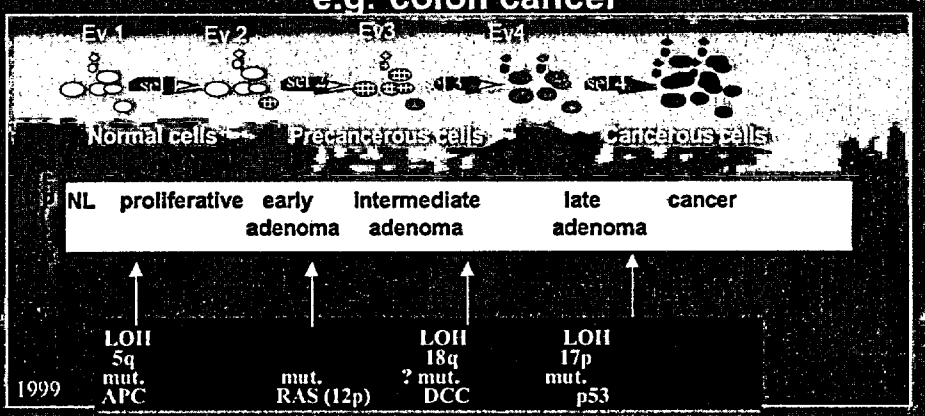
- Cells respond even very low radiation impacts.
- The response to IR involves activation of defense mechanisms, maintenance and death pathways.
- Cells react differently at different doses or dose rates of IR:
  - The IR response involves activation of signalling pathways and different gene families are activated.
  - At low doses and dose rates a multitude of parameters influence the cellular fate, whereas at high doses and doses rates cellular responses are more directly channelled towards survival, genomic instability and malignant transformation or cell death.

Conclusions

1. Recent data demonstrate that mammalian cells react differently at different levels of dose and dose rates of low LET radiation.
   —> DNA damage signalling, gene induction, DNA repair and apoptosis.
2. These differences in reactivity are consistent with practical thresholds observed at very low radiation doses (<20 mGy) but are [illegible].
   At low exposure levels cells appear to have more possibilities to cope with exogenous insults, and IR responses involve a wide ranging metabolic network. Cells are generally better protected at very low than at high dose levels ; and thus, human risks are likely to be lower than expected from LNT calculations.
3. [illegible] preferentially expressed at very low doses, are likely to influence dose-effect relationships for mutation induction and carcinogenesis of IR at low doses and dose-rates but the mechanisms involved and their actual quantitative impact need to be clarified.
4. [illegible] are very important for individual radiation responses but do not allow extrapolation to general population responses.

Multi-step carcinogenesis:

e.g. colon cancer



- Contribution of multiple interactions between the cell hosting a potentially oncogenic genetic event and its neighboring cells of the same type, the extracellular matrix
- The significance of epigenetic mechanisms is well documented (Baylin and Herman, 2000, Jones and Baylin, 2002)

Radiocarcinogenesis

- The conventional model acknowledged that, by a series of stages, stochastic alterations of the genome confer a selective advantage to a initiated cell, during carcinogenesis. These phenomena cannot be described by a linear process, during which successive genome damage of one cell accumulates at random.
- Carcinogenesis cannot be reduced to a series of mutations occurring in the same cell, it affects all aspects of genome function.

  against carcinogenic processes, and these must be successively overcome for carcinogenesis to occur.
- There are intracellular systems of proliferation control (suppressor genes), and mechanisms involving the death of initiated cells that tend to eliminate or prevent the proliferation of cells.
- At the whole body level, escape from the immune surveillance responsible for eliminating tumour cells is based on the selection of cells that are capable of escaping from it.
- Carcinogenesis may be facilitated by a reduction in immune defences

Radiocarcinogenic process

- **PREVIOUSLY:**
  - Initiated by specific genome lesions, each lesion adding itself to previous ones
  - Process considered to be a stochastic risk:
    - A rare event caused by the random occurrence of a lesion inside a target.
- **TODAY:**
  - Complex inducible reaction, dominated by intra- and intercellular signaling mechanisms, depending on oxidative stress.
- Carcinogenic mechanisms are sensitive to:
  - cellular microenvironment
  - interaction between initiated and healthy cells.
    - Reveal intricacy of genetic and epigenetic mechanisms

cell, tissue and body defences against cancerization

which has escaped to a saveguard mechanism: apoptotic response

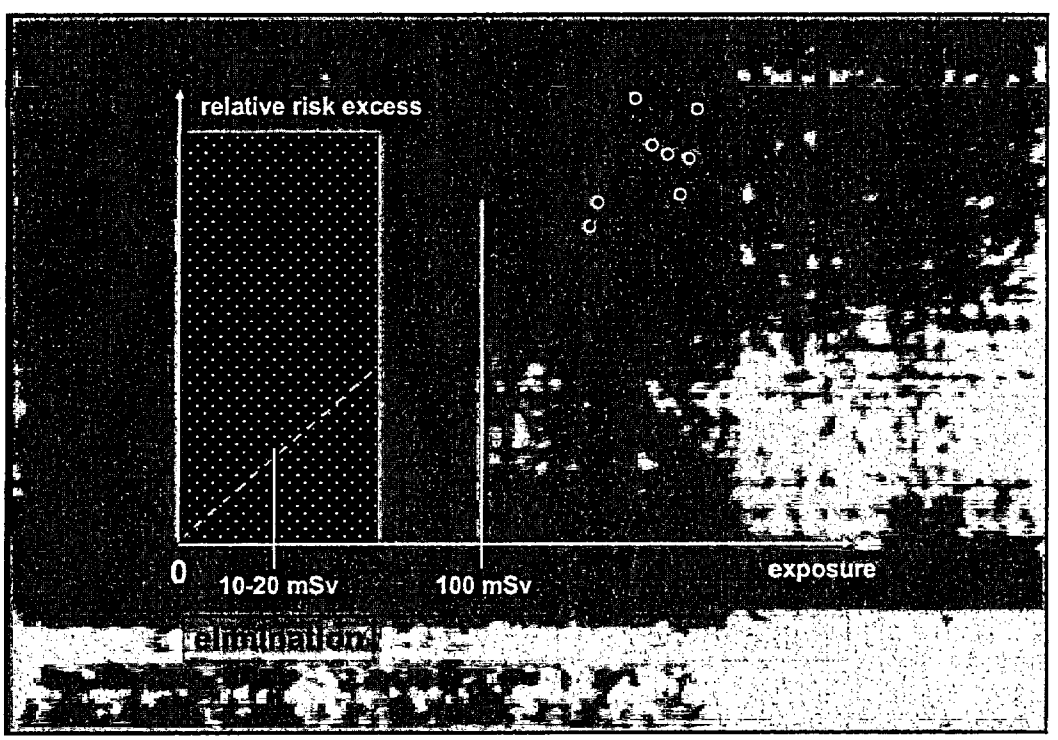
- secretion by neighboured cell and stroma of regulation factors, inhibitor of proliferation,

4 : exchange of signalisation and regulation molecules by intercellular gap junction,

ex. Healthy cells inhibit the development of potentially malignant clones.

Cell response

- The cell response therefore seems to depend on:
  - the dose,
  - the dose rate
  - the cell type,
  - and on the concentration of damaged cells



3-2. What to do with the problem?
ICRP well aware of the uncertainty

ICRP is very careful in using LNT, collective dose, and (cumulative dose)

Paragraph 29

"LNT is - - - to manage risk from radiation exposure"

Paragraph 146 - 147

"- in the case of low individual doses with wide geographical areas/long time scales, the use of collective dose for risk estimation - - is not reasonable and should be avoided"

Some differences between ICRP and BEIR VII

ICRP: pragmatic, realistic and conservative
LNT as a tool, not truth
supplemented with real data

BEIR VII: theoretical, idealistic and radical
LNT as science
based mainly on theory

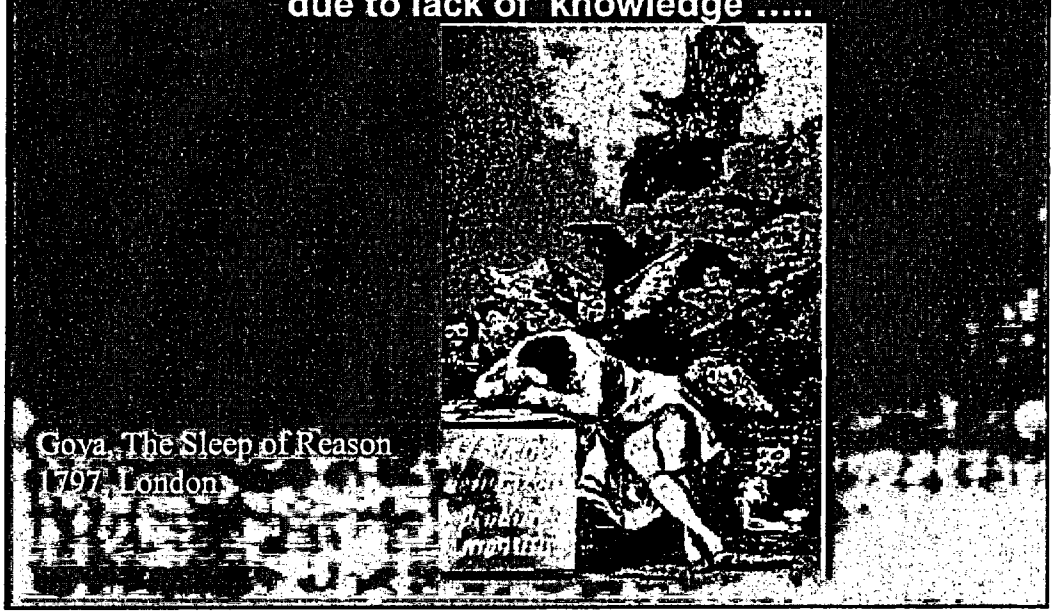
BEIR VII Report on page 30

"The Committee concludes that the current scientific evidence is consistent with the hypothesis that there is a linear, no-threshold dose-response relationship"

Conclusion

- While LNT may be useful for the administrative organisation of radioprotection, its use for assessing carcinogenic risks induced by low doses, such as those delivered by diagnostic radiology or the nuclear industry, is not based on valid scientific data.
- All the data show the lower effectiveness of low doses and dose rates. Moreover, the quantitative discrepancy between the results of the various epidemiological and animal experimental studies supports the view that there are several dose–effect relationships rather than only one,
- their parameters depend on the type of cancer, the type of ionising particle, radiation dose, dose rate, fractionation of irradiation, species, breeding line within the same species, target tissue, volume irradiated, age, and individual sensitivity factors.
- Epidemiological and biological data are compatible with the existence of a threshold but cannot today demonstrate its existence or assess its value (somewhere between 10 and 60 mSv)
- The concept of collective dose cannot be used for evaluating the cancer risk in a population.

In order to prevent radiation exposure from becoming unmanageable due to lack of knowledge .....



Goya, The Sleep of Reason
1797, London

Research and knowledge must come up with the most effective solution to deal with risk....

Francisco de Goya, ***El Parasol*** 1777

Joint report n° 2 of
the Academie des Sciences (Paris)
and the Academie Nationale de Medecine.

Dose-effect relationship and estimation of the carcinogenic effects of low doses of ionizing radiation.
M. Tubiana et al.

March 30, 2005, Editions Nucléon, pp. 1-94

Academie Nationale de Médecine

Académie des sciences

http://www.academie-medecine.fr/
http://www.academie-sciences.fr/

INTRODUCTORY STATEMENT BY ACNW CHAIRMAN
174th MEETING OF THE ADVISORY COMMITTEE ON NUCLEAR WASTE
NOVEMBER 13-16, 2006
ROCKVILLE, MARYLAND

WEDNESDAY, NOVEMBER 15, 2006 - 8:30 A.M.

THE MEETING WILL COME TO ORDER. THIS IS THE THIRD DAY OF THE 174th MEETING OF THE ADVISORY COMMITTEE ON NUCLEAR WASTE.

DURING TODAY'S MEETING, THE COMMITTEE WILL CONSIDER THE FOLLOWING:

1. DOSE EFFECT RELATIONSHIPS AND ESTIMATION OF THE CARCINOGENIC EFFECTS OF LOW DOSES OF IONIZING RADIATION

2. WHITE PAPER ON POTENTIAL ADVANCED FUEL CYCLES

3. DISCUSSION OF DRAFT ACNW LETTER REPORTS

THIS MEETING IS BEING CONDUCTED IN ACCORDANCE WITH THE PROVISIONS OF THE FEDERAL ADVISORY COMMITTEE ACT.

LATIF HAMDAN IS THE DESIGNATED FEDERAL OFFICIAL FOR TODAY'S INITIAL SESSION.

WE HAVE RECEIVED A REQUEST BY DR. THEODORE ROCKWELL FROM RADIATION, SCIENCE & HEALTH, INC., TO MAKE AN ORAL STATEMENT DURING TODAY'S SESSION.

SHOULD ANYONE ELSE WISH TO ADDRESS THE COMMITTEE, PLEASE MAKE YOUR WISHES KNOWN TO ONE OF THE COMMITTEE'S STAFF.

IT IS REQUESTED THAT THE SPEAKERS USE ONE OF THE MICROPHONES, IDENTIFY THEMSELVES, AND SPEAK WITH SUFFICIENT CLARITY AND VOLUME SO THAT THEY CAN BE READILY HEARD. IT IS ALSO REQUESTED THAT IF YOU HAVE CELL PHONES OR PAGERS, KINDLY TURN THEM OFF OR PLACE THEM ON MUTE. THANK YOU

INTRODUCTORY STATEMENT BY ACNW CHAIRMAN
174th MEETING OF THE ADVISORY COMMITTEE ON NUCLEAR WASTE
NOVEMBER 13-16, 2006
ROCKVILLE, MARYLAND

THURSDAY, NOVEMBER 16, 2006 - 8:30 A.M.

THE MEETING WILL COME TO ORDER. THIS IS THE FOURTH DAY OF THE 174th MEETING OF THE ADVISORY COMMITTEE ON NUCLEAR WASTE.

DURING TODAY'S MEETING, THE COMMITTEE WILL CONSIDER THE FOLLOWING:

1. PROPOSED REVISION TO REGULATORY GUIDE 1.112, CALCULATION OF RELEASES OF RADIOACTIVE MATERIALS IN GASEOUS AND LIQUID EFFLUENTS FROM LIGHT-WATER-COOLED REACTORS

2. PROPOSED REVISION TO REG GUIDE 4.15, QUALITY ASSURANCE FOR RADIOLOGICAL MONITORING PROGRAMS (INCEPTION THROUGH NORMAL OPERATIONS TO LICENSE TERMINATION) – EFFLUENT STREAMS AND THE ENVIRONMENT

3. DISCUSSION OF POTENTIAL ACNW LETTER REPORTS

4. DISCUSSION OF DRAFT ACNW LETTER REPORTS

5. MISCELLANEOUS

THIS MEETING IS BEING CONDUCTED IN ACCORDANCE WITH THE PROVISIONS OF THE FEDERAL ADVISORY COMMITTEE ACT.

MIKE LEE IS THE DESIGNATED FEDERAL OFFICIAL FOR TODAY'S INITIAL SESSION.

WE HAVE RECEIVED NO WRITTEN COMMENTS OR REQUESTS FOR TIME TO MAKE ORAL STATEMENTS FROM MEMBERS OF THE PUBLIC REGARDING TODAY'S SESSIONS. SHOULD ANYONE WISH TO ADDRESS THE COMMITTEE, PLEASE MAKE YOUR WISHES KNOWN TO ONE OF THE COMMITTEE'S STAFF.

IT IS REQUESTED THAT THE SPEAKERS USE ONE OF THE MICROPHONES, IDENTIFY THEMSELVES, AND SPEAK WITH SUFFICIENT CLARITY AND VOLUME SO THAT THEY CAN BE READILY HEARD. IT IS ALSO REQUESTED THAT IF YOU HAVE CELL PHONES OR PAGERS, KINDLY TURN THEM OFF OR PLACE THEM ON MUTE. THANK YOU

INTRODUCTORY STATEMENT BY ACNW CHAIRMAN
174th MEETING OF THE ADVISORY COMMITTEE ON NUCLEAR WASTE
NOVEMBER 13-16, 2006
ROCKVILLE, MARYLAND

MONDAY, NOVEMBER 13, 2006 - 10:00 A.M.

THE MEETING WILL COME TO ORDER. THIS IS THE FIRST DAY OF THE 174th MEETING OF THE ADVISORY COMMITTEE ON NUCLEAR WASTE.

DURING TODAY'S MEETING, THE COMMITTEE WILL CONSIDER THE FOLLOWING:

1. UPDATE ON STATUS OF SEISMIC DESIGN BASES AND METHODOLOGY: NRC PERSPECTIVE

2. RESULTS FROM THE LIQUID RADIOACTIVE RELEASE LESSONS LEARNED TASK FORCE

3. PREPARATION FOR MEETING WITH NRC COMMISSIONERS

THIS MEETING IS BEING CONDUCTED IN ACCORDANCE WITH THE PROVISIONS OF THE FEDERAL ADVISORY COMMITTEE ACT.

ANTONIO DIAS IS THE DESIGNATED FEDERAL OFFICIAL FOR TODAY'S SESSION.

WE HAVE RECEIVED NO WRITTEN COMMENTS OR REQUESTS FOR TIME TO MAKE ORAL STATEMENTS FROM MEMBERS OF THE PUBLIC REGARDING TODAY'S

SESSIONS. SHOULD ANYONE WISH TO ADDRESS THE COMMITTEE, PLEASE MAKE YOUR WISHES KNOWN TO ONE OF THE COMMITTEE'S STAFF.

IT IS REQUESTED THAT THE SPEAKERS USE ONE OF THE MICROPHONES, IDENTIFY THEMSELVES, AND SPEAK WITH SUFFICIENT CLARITY AND VOLUME SO THAT THEY CAN BE READILY HEARD. IT IS ALSO REQUESTED THAT IF YOU HAVE CELL PHONES OR PAGERS, KINDLY TURN THEM OFF OR PLACE THEM ON MUTE.

I WILL BEGIN WITH SOME ITEMS OF CURRENT INTEREST.

MR. CHRISTOPHER BROWN JOINED THE ACNW IN OCTOBER. HE BEGAN HIS EMPLOYMENT AT THE NRC IN 1996 AS A MECHANICAL ENGINEER IN THE DIVISION OF INDUSTRIAL AND MEDICAL NUCLEAR SAFETY IN THE OFFICE OF NUCLEAR MATERIAL SAFETY AND SAFEGUARDS, WHERE HE PERFORMED SEALED SOURCE AND DEVICE REVIEWS. IN 1998, HE JOINED THE SPENT FUEL PROJECT OFFICE AS A MATERIALS ENGINEER WHERE HE PERFORMED MATERIALS AND CONTAINMENT REVIEWS FOR DRY CASK STORAGE SYSTEMS AND TRANSPORTATION PACKAGES. MR. BROWN HAS ALSO HAD THE OPPORTUNITY TO ROTATE TO THE DIVISION OF REACTOR SAFETY SYSTEMS IN THE OFFICE OF NUCLEAR REACTOR REGULATION TO FURTHER DEVELOP HIS EXPERTISE IN THE FUELS AREA. MR. BROWN HOLDS A B.S. IN ENGINEERING PHYSICS FROM MORGAN STATE UNIVERSITY AND A M.S. IN MATERIALS SCIENCE AND ENGINEERING FROM UNIVERSITY OF MARYLAND.

INTRODUCTORY STATEMENT BY ACNW CHAIRMAN
174th MEETING OF THE ADVISORY COMMITTEE ON NUCLEAR WASTE
NOVEMBER 13-16, 2006
ROCKVILLE, MARYLAND

TUESDAY, NOVEMBER 14, 2006 - 8:30 A.M.

THE MEETING WILL COME TO ORDER. THIS IS THE SECOND DAY OF THE 174th MEETING OF THE ADVISORY COMMITTEE ON NUCLEAR WASTE.

DURING TODAY'S MEETING, THE COMMITTEE WILL CONDUCT A WORKING GROUP MEETING ON DECOMMISSIONING LESSONS LEARNED.

THIS MEETING IS BEING CONDUCTED IN ACCORDANCE WITH THE PROVISIONS OF THE FEDERAL ADVISORY COMMITTEE ACT.

DEREK WIDMAYER IS THE DESIGNATED FEDERAL OFFICIAL FOR TODAY'S SESSION.

WE HAVE RECEIVED NO WRITTEN COMMENTS OR REQUESTS FOR TIME TO MAKE ORAL STATEMENTS FROM MEMBERS OF THE PUBLIC REGARDING TODAY'S SESSIONS. SHOULD ANYONE WISH TO ADDRESS THE COMMITTEE, PLEASE MAKE YOUR WISHES KNOWN TO ONE OF THE COMMITTEE'S STAFF.

IT IS REQUESTED THAT THE SPEAKERS USE ONE OF THE MICROPHONES, IDENTIFY THEMSELVES, AND SPEAK WITH SUFFICIENT CLARITY AND VOLUME SO THAT THEY CAN BE READILY HEARD. IT IS ALSO REQUESTED THAT IF YOU HAVE CELL PHONES OR PAGERS, KINDLY TURN THEM OFF OR PLACE THEM ON MUTE. THANK YOU.

SPENT NUCLEAR REACTOR FUEL REPROCESSING

HISTORICAL REVIEW AND FORWARD LOOK

CONTRIBUTORS:

RAYMOND WYMER, Consultant
HOWARD LARSON, Consultant
LAWRENCE TAVLARIDES, Consultant
JOHN FLACK, ACNW Staff

ACNW LEAD MEMBER: ALLEN CROFF

November 15, 2006

OVERVIEW

- INTRODUCTION – J. FLACK
- HISTORICAL PERSPECTIVE – R. WYMER
- UREX+1A PROCESS STREAM ANALYSES – L. TAVLARIDES
- PLANT DESIGN AND FACILITIES –R. WYMER
- REGULATORY CONNECTION – J. FLACK
- ISSUES AND DISCUSSION – R. WYMER

COMMISSION SRM (FEBRUARY 7, 2007)

IN RESPONSE TO ACNW FY 2006-2007 ACTION PLAN

- "THE ACNW SHOULD REMAIN ABREAST OF INDUSTRY, TECHNICAL AND LEGAL DEVELOPMENTS IN THE AREAS OF SPENT FUEL STORAGE, DISPOSAL AND REPROCESSING TO ENSURE THAT THE MEMBERS WILL BE READY TO PROVIDE ADVICE IN THESE AREAS, SHOULD THE NEED ARISE."

- "AN IMPORTANT DESIGN CRITERION FOR ANY NEW REPROCESSING EFFORT WILL BE THAT DECOMMISSIONING COSTS BE MANAGEABLE."

CURRENT ACNW INITIATIVES

ACTION PLAN (TIER 2) FUEL CYCLE FACILITY:

- BECOME FAMILIAR WITH FUEL CYCLES FOR ADVANCED REACTOR SYSTEMS
- KEEP INFORMED OF TECHNICAL DEVELOPMENTS
- IN 2007, ACQUIRE GREATER UNDERSTANDING THROUGH SITE VISITS

DOE CURRENT PLANS

- ENGINEERING SCALE DEMONSTRATION (ESD)
  (DISCONTINUED AT THE PRESENT TIME)
- ADVANCED FUEL CYCLE FACILITY
  (ENGINEERING SCALE DEMONSTRATION)
- CONSOLIDATED FUEL TREATMENT CENTER (CFTC) (COMMERCIAL SCALE),-
  RFP FY 2007
- ADVANCED BURNER REACTOR
  (COMMERCIAL SCALE)

PRESENTATION BY Dr. R.G. Wymer

CONSULTANT ON THE NUCLEAR FUEL CYCLE

WHITE PAPER CONTENTS SUMMARY

WHITE PAPER CONTENTS-1

- HISTORICAL EXPERIENCE
- INTERNATIONAL FUEL CYCLE INITIATIVES
- DOE RECYCLE PROGRAM STATUS AND FLOWSHEETS
- PLANT DESIGN AND OPERATIONAL FEATURES

WHITE PAPER CONTENTS-2

- TECHNICAL, SAFETY, LICENSING, AND REGULATORY ISSUES FOR RECYCLE FACILITIES
- APPROACHES FOR ENSURING OPERATIONAL SAFETY
- PATH FORWARD

EARLY U.S. REPROCESSING PLANTS

Pu PRODUCTION AND NAVAL FUEL

- HANFORD - PRODUCTION
- SAVANNAH RIVER - PRODUCTION
- IDAHO FALLS – NAVAL FUEL

COMMERCIAL SPENT FUEL

- WEST VALLEY - OPERATED
- ALLIED GENERAL NUCLEAR FUELS (BARNWELL) – NEVER OPERATED
- GE MORRIS PLANT – NEVER OPERATED

PRINCIPLE FOREIGN REPROCESSERS

- FRANCE
- UNITED KINGDOM
- RUSSIA
- JAPAN
- CHINA – DEFENSE ONLY
- INDIA – DEFENSE AND CIVIL

FOREIGN REPROCESSING CIVIL CAPACITY, MTHM/YR

LWR fuel

France, La Hague:1700
UK, Sellafield (THORP): 900
Russia, Ozersk (Mayak): 400
Japan,
Tokai: 14;
Rokkasho: 800
Total approx. 3814

Other nuclear fuels

UK, Sellafield: 1500
India: 275
Total approx. 1775
TOTAL CIVIL CAPACITY: 5589

PROLIFERATION-RESISTANT FUEL CYCLE INITIATIVES

- INTERNATIONAL NUCLEAR FUEL CYCLE EVALUATION (INFCE:1977-1980)
- U.S. GLOBAL NUCLEAR ENERGY PARTNERSHIP (GNEP)
- RUSSIAN GLOBAL NUCLEAR INFRASTRUCTURE (GNI)

INFCE: COVERAGE OF THE STUDY

- NUCLEAR FUEL CYCLE ASSESSMENT
- IMPROVE Pu FUELS AVAILABILITY TO DEVELOPING NATIONS
- SPENT NUCLEAR FUEL STORAGE
- IMPROVED NUCLEAR SAFEGUARDS
- ALTERNATIVES TO A Pu AND HEU NUCLEAR ECONOMY

GLOBAL NUCLEAR ENERGY PARTNERSHIP (GNEP) GOALS

- EXPAND DOMESTIC USE OF NUCLEAR POWER
- DEMONSTRATE PROLIFERATION-RESISTANT FUEL CYCLES
- MINIMIZE NUCLEAR WASTE (OBVIATE NEAR-TERM NEED FOR ADDITIONAL GEOLOGIC REPOSITORIES)
- DEVELOP AND DEMONSTRATE ADVANCED BURNER REACTORS
- ESTABLISH LEASE-AND-RETURN FUEL CYCLE SERVICES
- DEMONSTRATE SMALL-SCALE REACTORS
- DESIGN NUCLEAR SAFEGUARDS INTO FACILITIES AND REACTORS

RUSSIAN GLOBAL NUCLEAR INFRASTRUCTURE INITIATIVE (GNI)

- ALTERNATIVE TO GNEP?
- ESTABLISH FULL-SERVICE INTERNATIONAL NUCLEAR CENTERS
- NUCLEAR CENTERS ONLY IN NUCLEAR WEAPONS STATES
- RUSSIAN PILOT ENRICHMENT CENTER AT ANGARST IN SIBERIA UNDER IAEA SUPERVISION
- SHAREHOLDING STRUCTURE FOR COUNTRIES INVOLVED IN CENTER

GENERATION IV INTERNATIONAL FORUM (MAY 2001)

GOAL: DEVELOP NEXT GENERATION NUCLEAR ENERGY SYSTEMS

APPROACH: DEVELOPMENT OF FIVE DIFFERENT REACTOR SYSTEMS:

- PWR– EVOLUTIONARY DEVELOPMENT
- BWR– EVOLUTIONARY DEVELOPMENT
- FBR– MOLTEN METAL AND GAS COOLED
- HTGR– PEBBLE BED
- MSR

LWR FUEL REPROCESSING

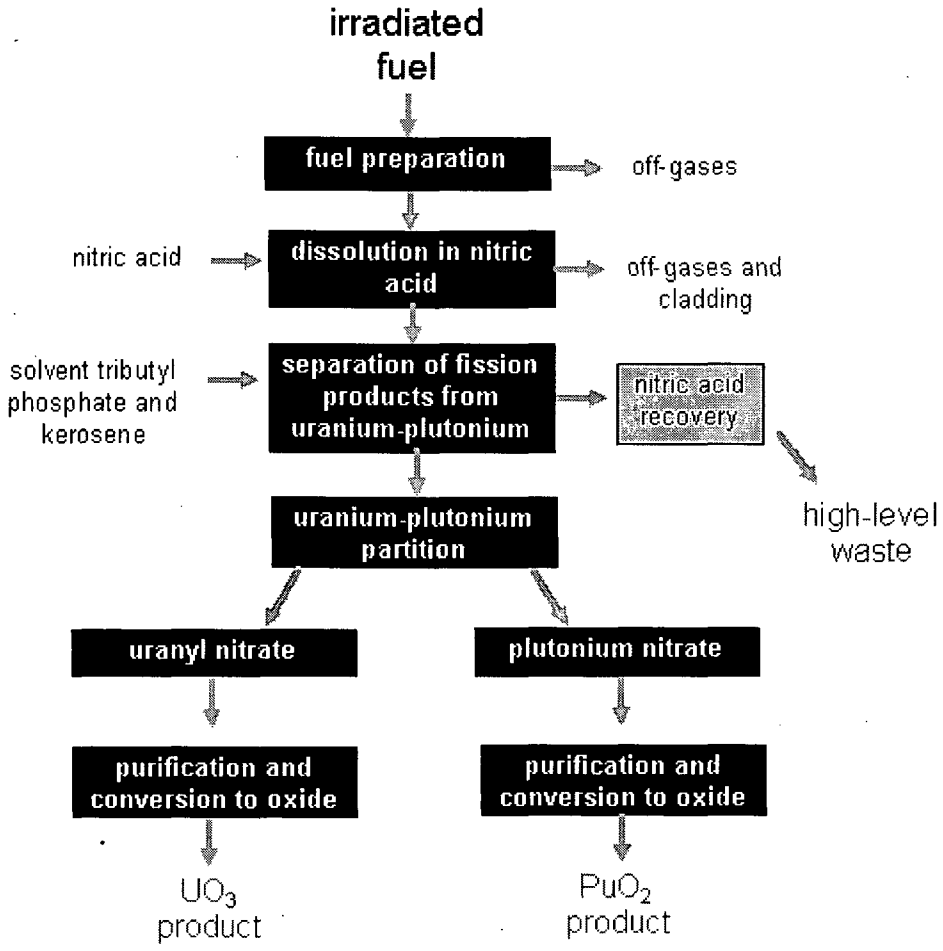
CONVENTIONAL

- PUREX

PROLIFERATION RESISTANT

- UREX ALTERNATIVES (U.S.)
- GANEX (FRENCH)

PUREX PROCESS

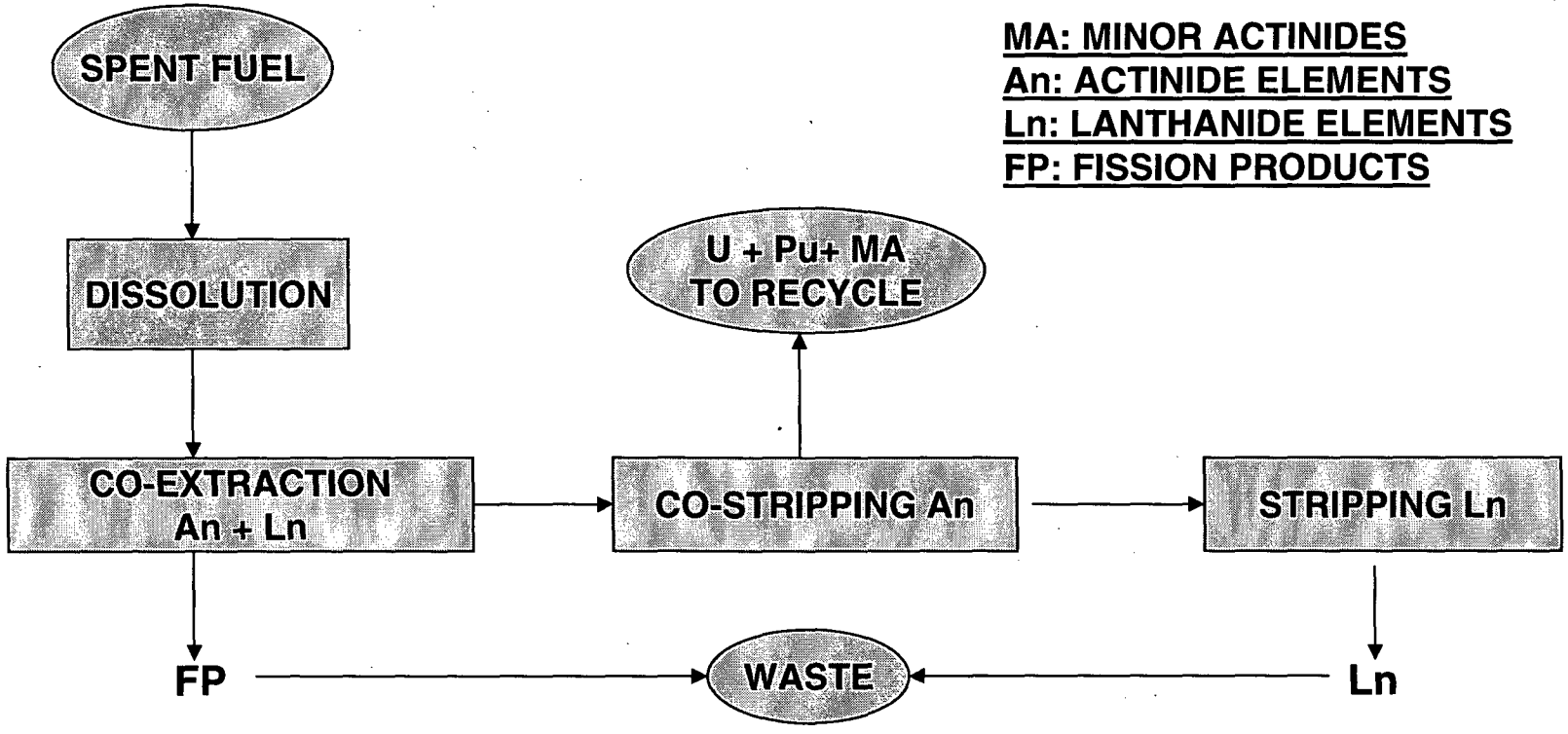


UREX PROCESS ALTERNATIVES

UREX PROCESS	PRODUCT # 1	PRODUCT # 2	PRODUCT # 3	PRODUCT # 4	PRODUCT # 5	PRODUCT # 6	PRODUCT # 7
UREX +1	U	Tc	Cs/Sr	TRU + Ln	FP		
UREX + 1A	U	Tc	Cs/Sr	TRU	ALL OTHER FP		
UREX + 2	U	Tc	Cs/Sr	Np + Pu	Am + Cm + Ln	ALL OTHER FP	
UREX + 3	U	Tc	Cs/Sr	Np + Pu	Am + Cm + ALL OTHER FP	ALL OTHER FP	
UREX + 4	U	Tc	Cs/Sr	Np + Pu	Am	Cm	ALL OTHER FP

FRENCH GANEX PROCESS

ALSO CALLED A COEX PROCESS



PRESENTATION BY DR. LAWRENCE TAVLARIDES

PROFESSOR, BIOMEDICAL AND CHEMICAL ENGINEERING

SYRACUSE UNIVERSITY
lltavlar@syr.edu

UREX +1A FLOWSHEETS AND EQUIPMENT

UREX+1A PROCESS STREAMS INPUT

- Case Studies to evaluate:
  - 45 GWD/MTIHM – 5 yr and 30 yr cool-down time
  - 60 GWD/MTIHM – 5 yr and 30 yr cool-down time
  - 1 MTHM/Day – engineering scale limit

- Flowsheet Analyses Preparation:
  - ORIGEN BURN UP calculations – ORNL: J. E. Ruston, I. C. Guald, B.D. Murphy

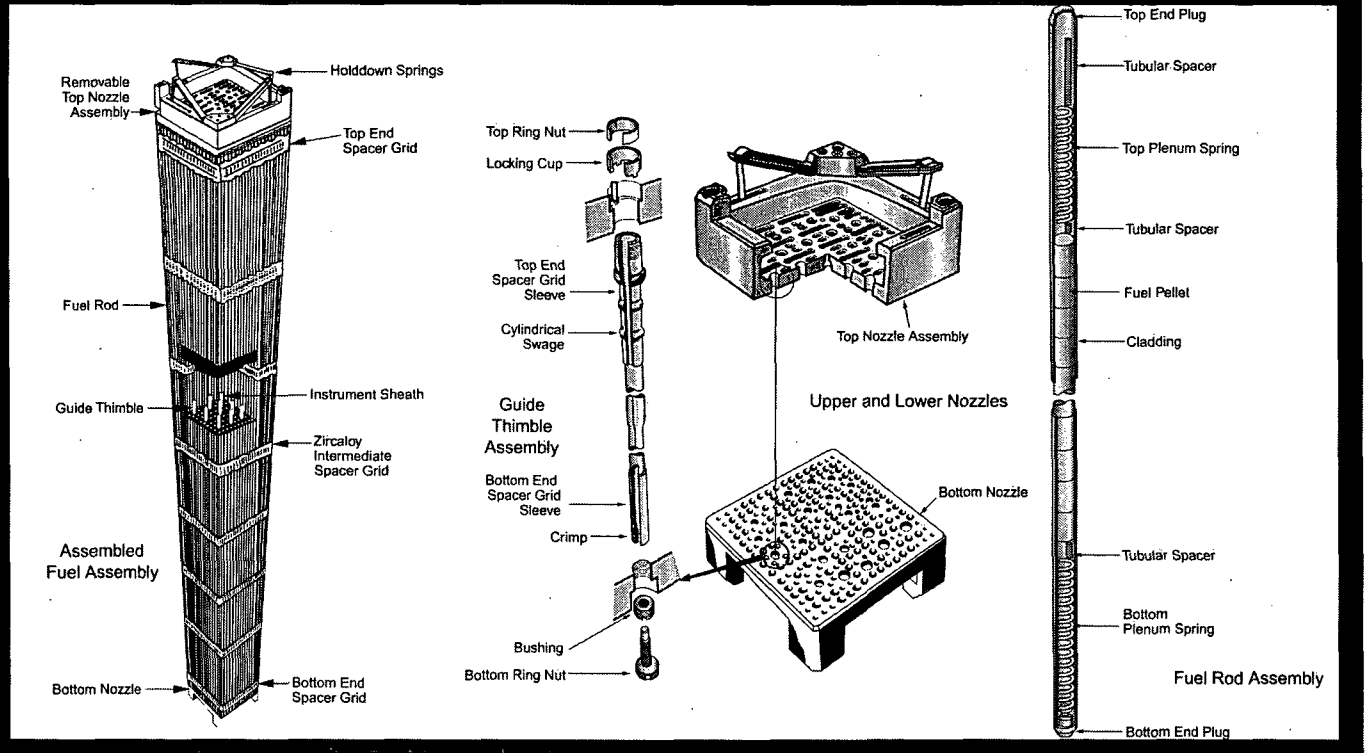
- Typical PWR Assembly, Kg

U	461.4	
UO_2	523.4	
Zircaloy 4	108.4	(cladding and guide tubes)
SS 304	17.1	(end fittings)
SS 302	1.9	(plenum spring)
Inconel	5.9	(grid spacers)
Nicrobraze	1.2	(brazing alloy)

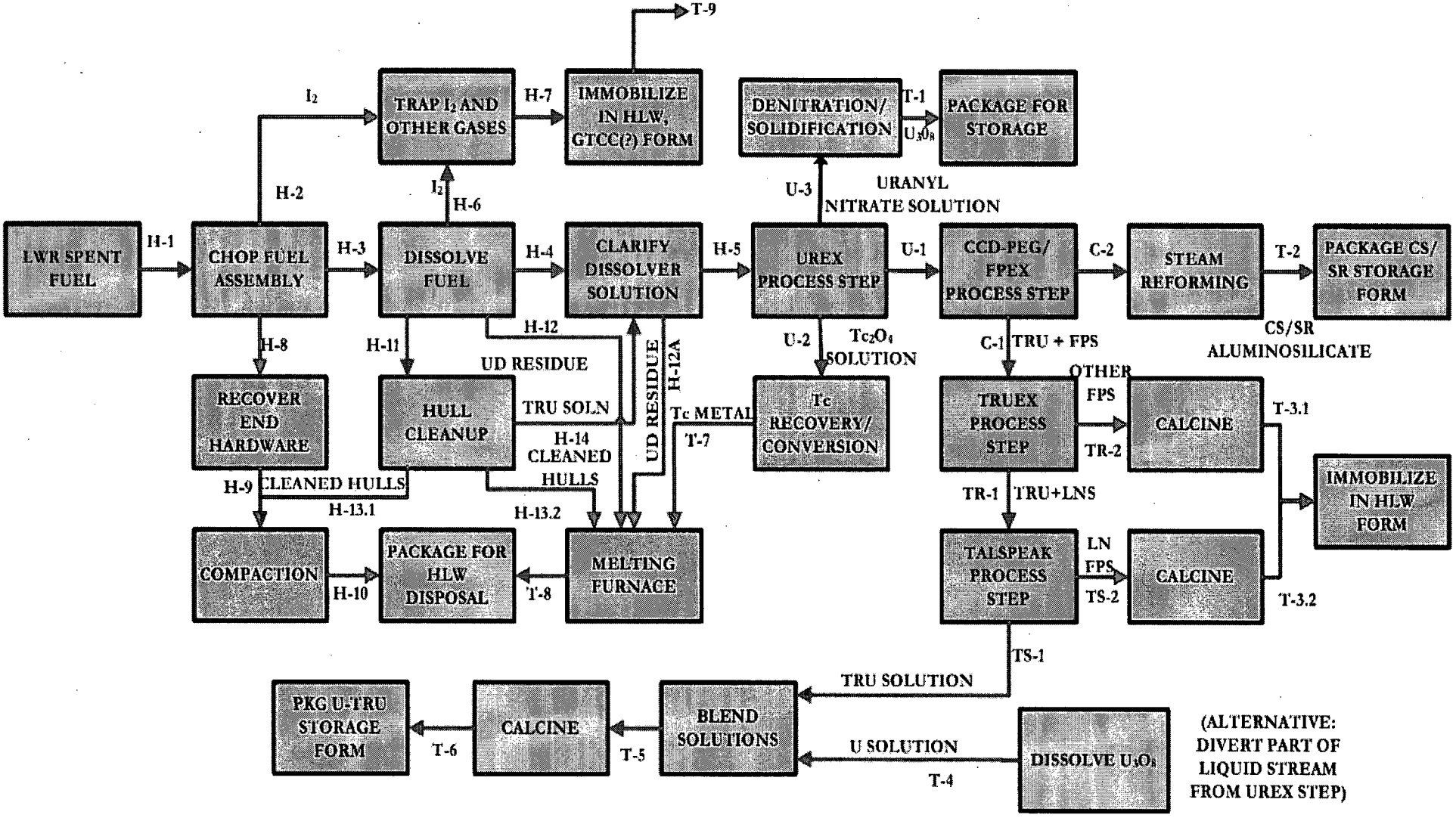
TOTAL HARDWARE: 134.5 Kg

PRESSURIZED WATER REACTOR FUEL

MAJOR COMPONENTS OF PWR FUEL ASSEMBLY



UREX+1A PROCESS: HEAD END, CENTRAL AND TAIL END UNIT OPERATIONS[d]



[d] *Expert Panel Report on Scaling Engineering Scale Demonstration GNEP, US DOE, 07-28-06*

Elemental Feed and Curie Composition

ORIGEN DATA
60 GWD/MT IHM
5 Year Cool-Down

Fuel Metal Basis	Weight (g)	Radiation (Curies)
Heavy Metal*+	1.00E+06	-
End Hardware*	1.82E+05	-
Hulls*	1.82E+05	-
Gases		
I	3.21E+02	5.67E-02
Kr	4.94E+01	1.10E+04
H	6.61E-02	6.39E+02
C	2.06E-01	9.20E-01
U	9.23E+05	5.95E+00
TRU		
Np	9.70E+02	?
Pu	1.17E+04	1.71E+05
Am	8.21E+02	1.79E+03
Cm	1.40E+03	1.05E+04

**Taken from Laidler, J. J., "The GMEP Partnership" ACNW July 20, 2006.*
** End Hardware weight split between hardware and hulls, values adjusted to 1 MTIHM*
+ Does not include oxygen

Elemental Feed and Curie Composition (Cont.)

Fuel Metal Basis	Weight (g)	Radiation (Curies)
Cs	4.64E+03	1.78E+05
Sr	1.46E+03	1.18E+05
Tc	1.33E+03	2.28E+01
Ru	4.00E+03	2.72E+04
Rare Earths		
Ce	4.45E+03	1.67E+04
Pr	2.21E-04	1.69E+04
Nd	5.96E+03	?
Pm	5.99E+01	5.56E+04
Sm	1.42E+03	6.39E+02
Eu	2.64E+02	1.24E+04
Gd	2.93E+02	1.89E-01
Se	9.70E+01	1.20E+00
Sn	9.42E+01	6.90E+00
Ba	3.03E+03	1.62E+05
Zr*	6.04E+03	2.09E+00
Rb	4.30E+02	-

*FP and Cladding

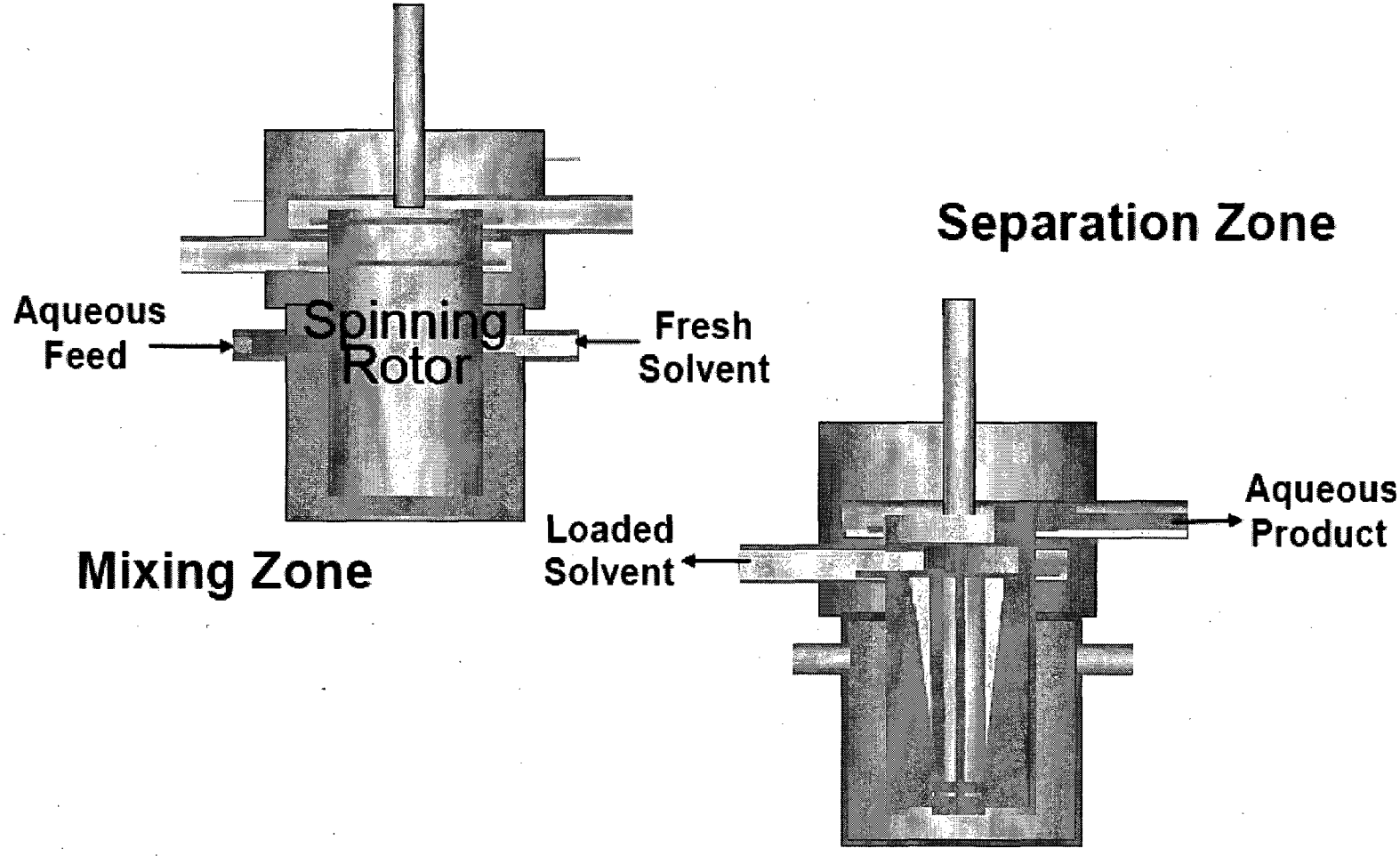
UREX, CCD-PEG, TRUEX, TALSPEAK – FLOW SHEETS[b,c]

- **THE FLOW SHEETS INCLUDE OPERATIONS FOR OFF-PRODUCT RECYCLE, SOLVENT WASH AND SOLVENT RECYCLE**

[b]Vandegrift, G. F. et al., "Designing and Demonstration of the UREX + Process Using Spent Nuclear Fuel", Presentation at ATLANTE '04 Nimes, France, June 21-24, 2004.

[c]Pereira, C. et al., "Primary Results of the Lab-scale Demonstration of the UREX+1a Process Using Spent Nuclear Fuel", Presented at AIChE National Meeting, November 3, 2005

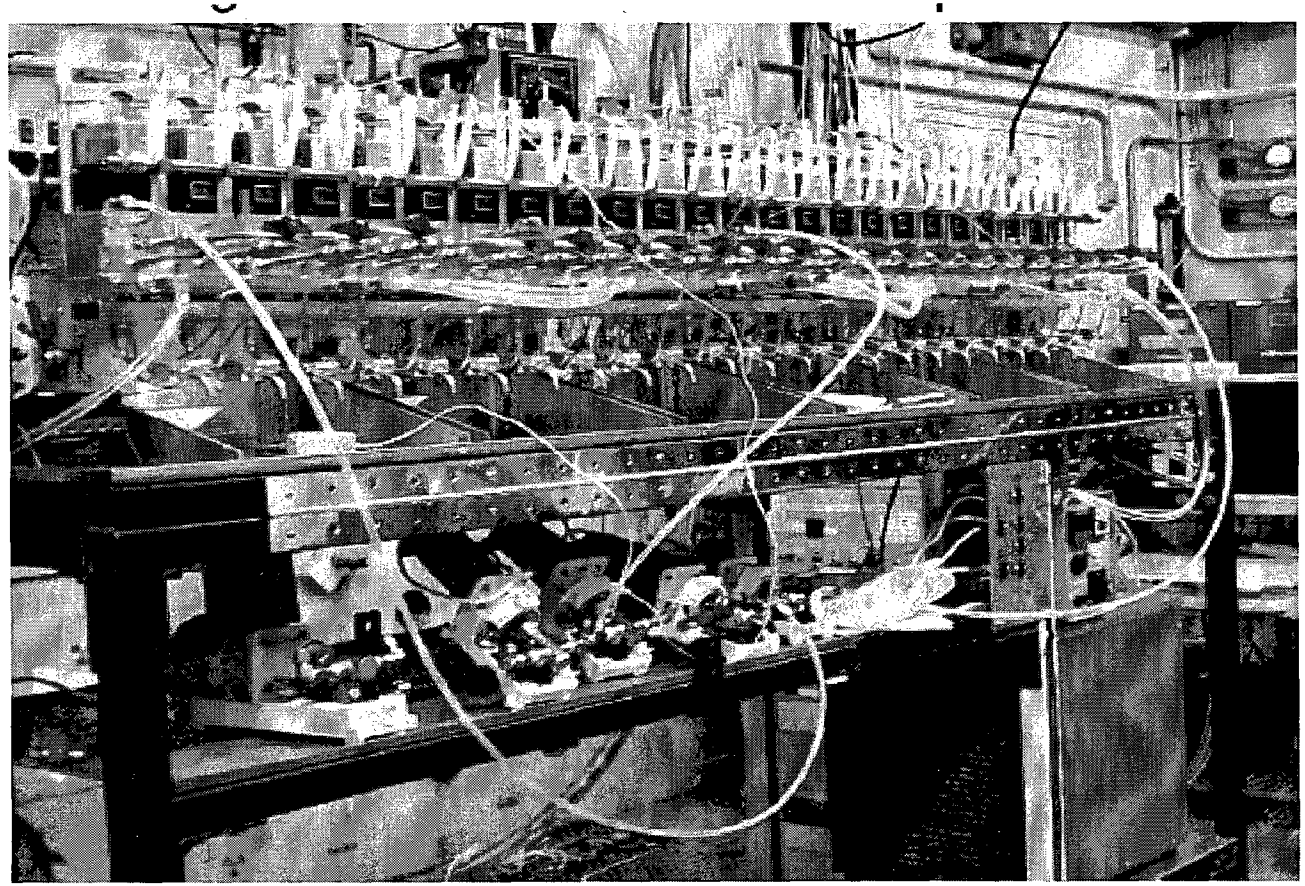
CENTRIFUGAL CONTACTORS[e]



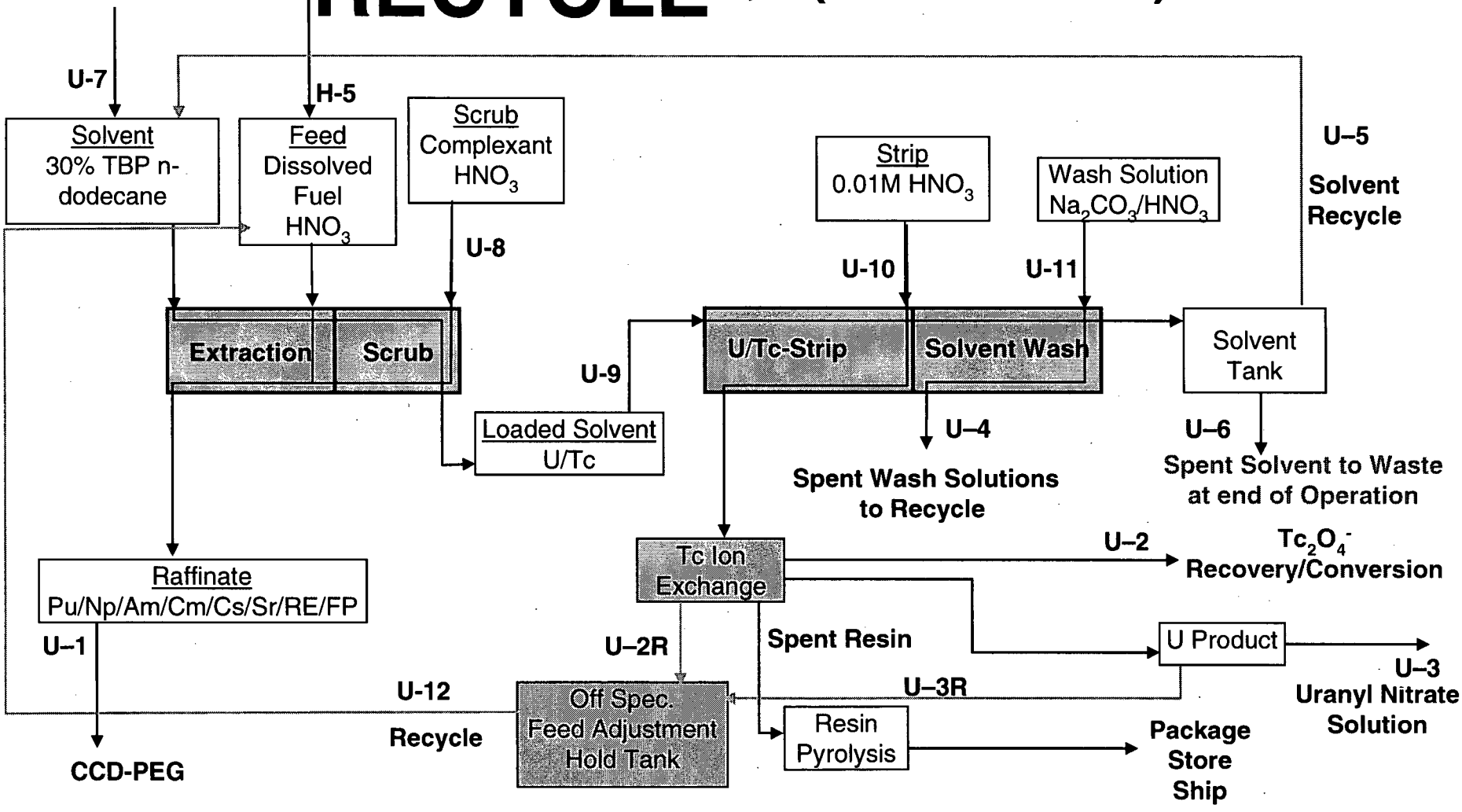
[e] *Candido Pereira et al., "Preliminary results of lab-scale demonstration of the UREX+1a process using spent nuclear fuel" 2005 AIChE National Meeting November 3, 2005.*

UREX+1a PROCESS EQUIPMENT[e]

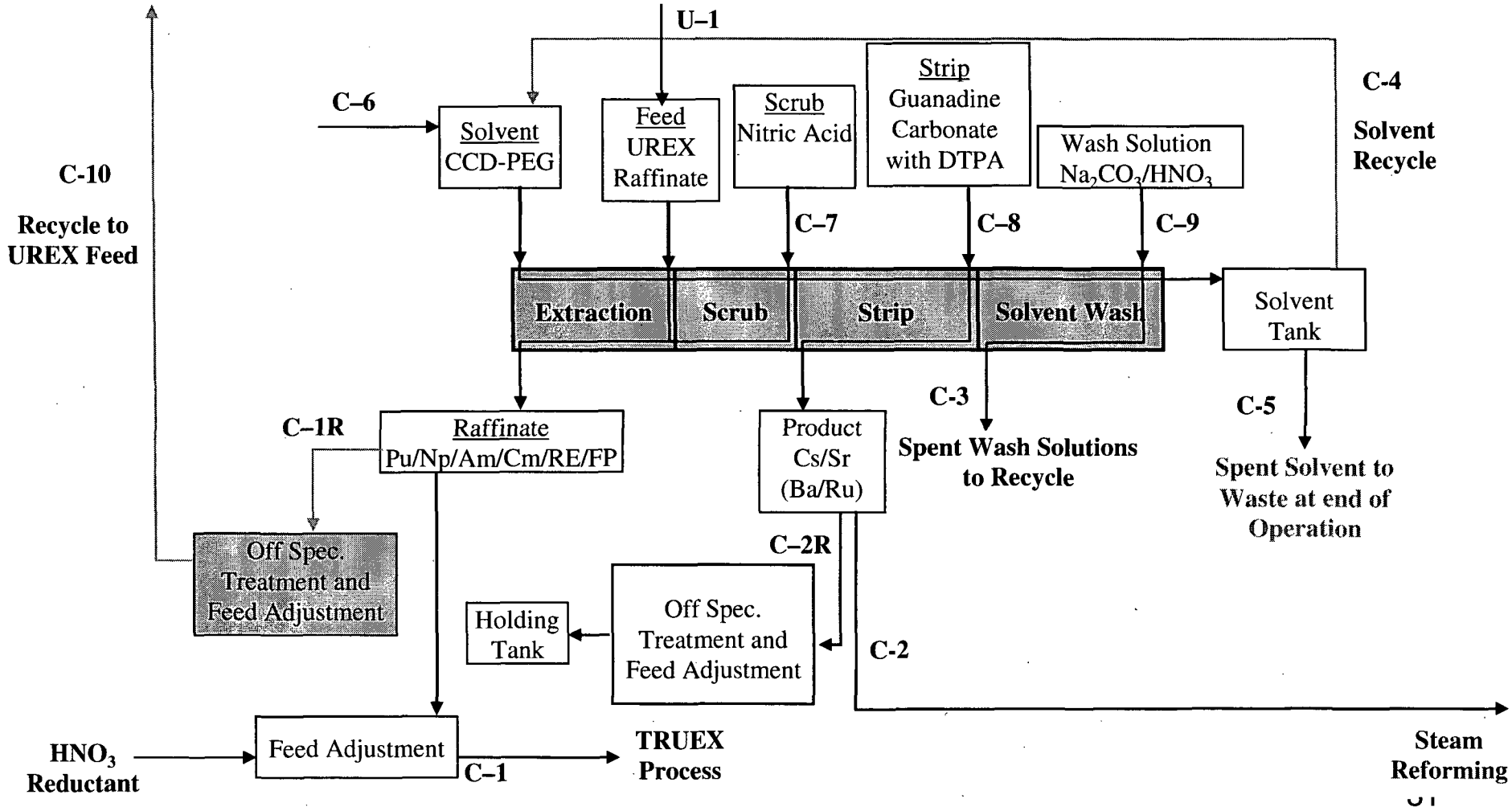
- 2-cm Centrifugal Contactor Bank before placement in hot cell



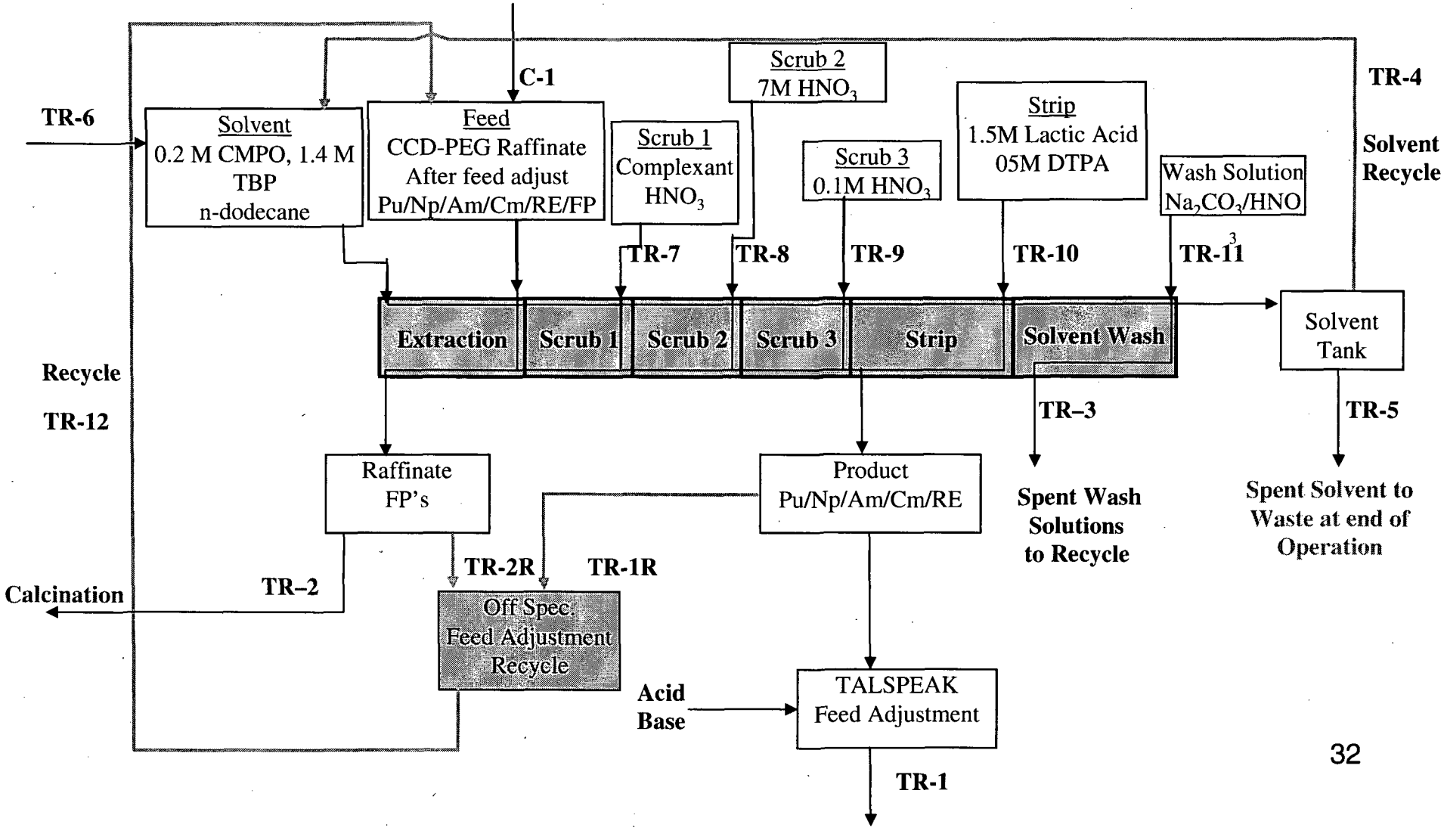
UREX + 1A PROCESS WITH RECYCLE[b,c] (FIRST STEP)



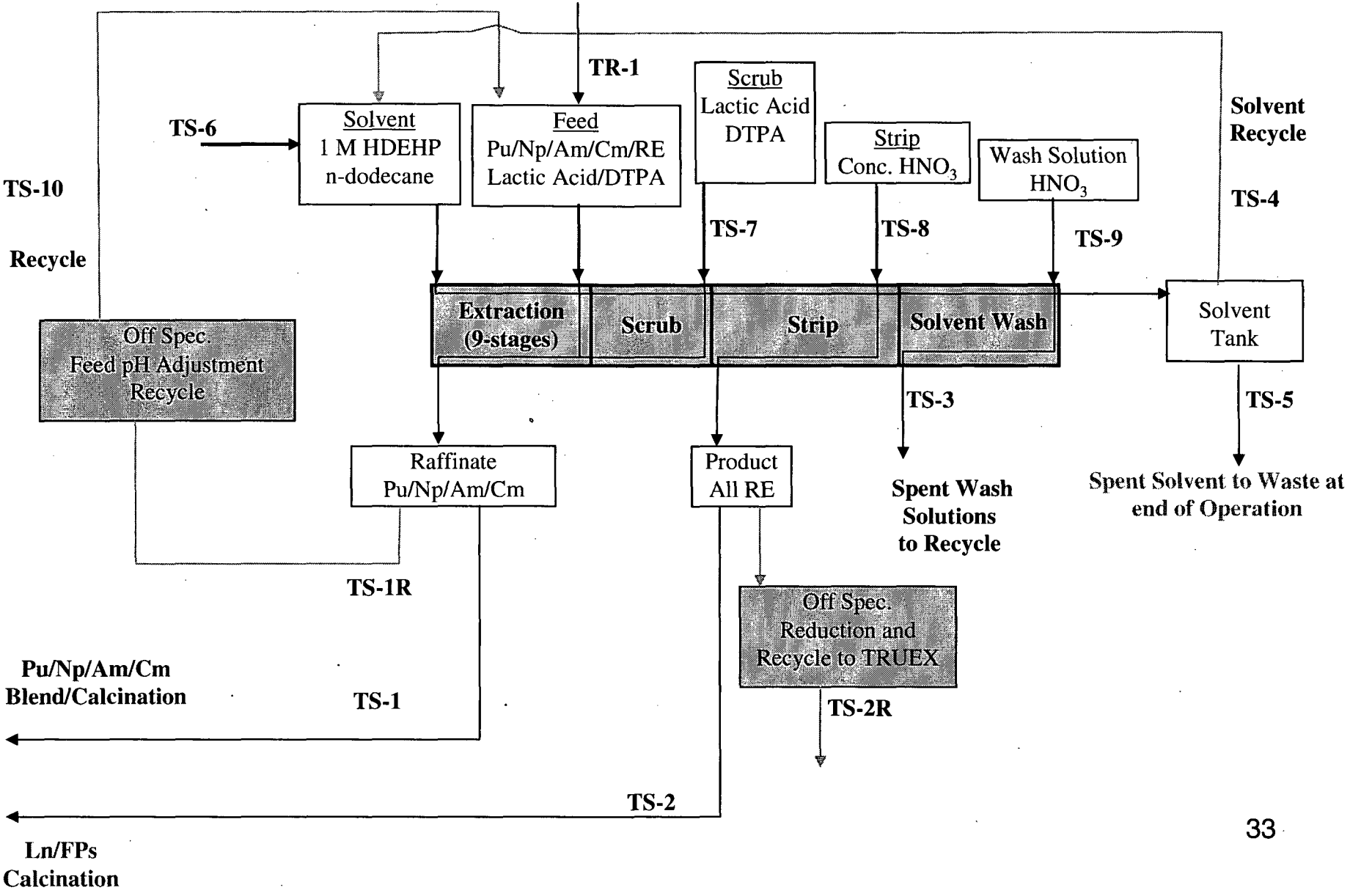
UREX + 1A CCD-PEG PROCESS STEP WITH RECYCLE[b,c]



UREX + 1A TRUEX PROCESS STEP WITH RECYCLE[b,c]



UREX + 1A TALSPEAK PROCESS STEP WITH RECYCLE[b,c]



UREX+1A PROCESS WASTE AND EFFLUENT STREAMS

	Waste Form /Product	Disposition
HEAD END	Hdw-Hull Compacted	HLW, GTCC(?)
	UDS (fuel dissolution, etc)	HLW
	I-129/Crystalline	HLW, GTCC(?)
	Kr-85/compressed gas, C-14	Temporary Decay Storage
	H-3	Temporary Decay Storage/Release
	Tc Metallic Waste	HLW

UREX+1A PROCESS WASTE AND EFFLUENT STREAMS (Cont.)

CENTRAL	Cs/Sr (waste form produced by steam reforming process)	HLW cooled Binsets
	TRUEX/TALSPEAK – FP's (Zircaloy metal matrix or calcine)	HLW
	Spent Solvents (TBP, CCD-PEG, CMPO, HDEHP, AHA, n-dodecane)	Incineration (?)
	Vessel off gases	Recycle to Head End Treatment
	Off –Gas control systems secondary waste	Class C(?)
TAIL END	Pkg U-TRU product	HLW storage for fuel

UREX + 1A FLOWSHEET ATTRIBUTES FOR REGULATORY CONSIDERATION

- AMOUNTS AND TYPES OF GASEOUS EFFLUENTS
- AMOUNTS AND TYPES OF LIQUID WASTES
  - HLW FOR VITRIFICATION – FISSION PRODUCTS
  - LAW FOR CEMENTATION AND DRUMMING
  - SOLVENTS -- INCINERATE
- AMOUNTS AND TYPES OF SOLID WASTES
  - EQUIPMENT
  - RESINS
  - GTCC MISC.
- INTERIM PACKAGING AND DISPOSAL OF ^{137}Cs AND ^{90}Sr
- INTERIM PACKAGING AND STORAGE OF ACTINIDES

REPROCESSING PLANT DESIGN EXPERIENCE, FACILITIES, LICENSING AND OPERATIONS INFORMATION PROVIDED BY

HOWARD LARSON

President and General Manager of the AGNS plant (BNFP) in Barnwell, SC;
Former Sr. Staff Engineer and Team Leader ACRS/ACNW
U.S. Nuclear Regulatory Commission

REPROCESSING PLANT SITING CONSIDERATIONS

- Proximity to reactors
- Geology
- Hydrology
- Seismology
- Climatology
- Flooding
- Topography
- Demographics
- Agriculture
- Area Industry

REPROCESSING PLANT MAJOR FACILITIES

1. FUEL RECEIVING AND INTERIM STORAGE
2. SEPARATIONS PROCESSES
3. URANIUM PRODUCT PREPARATION
4. PLUTONIUM PRODUCT PREPARATION
5. WASTE STORAGE AND SOLIDIFICATION

DESIGN CONSIDERATIONS

- ROUTINE ENVIRONMENTAL RADIOACTIVITY RELEASES ONLY BY AIR PATHWAYS
- CORROSION RESISTANT PROCESS EQUIPMENT
- CONFINEMENT INTEGRITY AGAINST EARTHQUAKES AND TORNADOES
- REMOTELY MAINTAINED HIGH-RADIATION CELLS
- PLANT AREA ACCESS CONTROLLED BY RADIATION ZONES
- CRITICALITY CONTROL

TYPICAL REPROCESSING PLANT EFFLUENT WASTE STREAMS-1

EFFLUENT GASES

- ^{85}Kr (DISSOLVER OFF-GAS; UNTREATED IN THE PAST)
- ^{129}I (DISSOLVER OFF-GAS; REMOVED IN THE PAST)
- ^{14}C (as CO_2) (DISSOLVER OFF-GAS; UNTREATED IN THE PAST)
- TRITIUM (RELEASED IN THE PAST DURING DECLADDING)

SOLIDS AND LIQUIDS

- HLW – SOME VITRIFIED
- LAW – SOLVENT CLEANUP; IX RESINS
- MISCELLANEOUS

TYPICAL REPROCESSING PLANT EFFLUENT WASTE STREAMS-2

SOLIDS AND LIQUIDS - CONTINUED

- FUEL CLADDING AND HARDWARE
- STABILIZED LIQUID WASTES - CONCRETE
- ANALYTICAL WASTES
- EQUIPMENT
- GTCC WASTE (TRU) STABILIZED FOR DISPOSAL

LIQUID HIGH-LEVEL WASTE (HLW) STORAGE

- HIGHLY RADIOACTIVE: GENERATES HEAT
- STORED IN LARGE, COOLED UNDERGROUND TANKS UNTIL SHORT-LIVED RADIONUCLIDES HAVE DECAYED

MANAGEING PLANT WASTES

WASTE TREATMENT

- WASTES CONVERTED TO A SOLID FOR TRANSPORT AND DISPOSAL
- HLW TYPICALLY CONTAINED IN BOROSILICATE GLASS
- OTHER LIQUID WASTES IMMOBILIZED WITH CEMENT; LAW SOLIDS ARE DRUMMED

WASTE DISPOSAL

- HLW, CLADDING HULLS, TRU WASTES: IN A GEOLOGIC REPOSITORY
- OTHER WASTES: TYPICALLY IN SURFACE TRENCHES
- ^{129}I HAS NO INERT, STABLE CHEMICAL FORM SUITABLE FOR LONG-TERM DISPOSAL

OPERATING PERSONNEL FOR REPROCESSING PLANTS

- OPERATOR TRAINING AND LICENSING
- MAJOR OPERATOR CATEGORIES
  - MANIPULATOR
  - CHEMICAL
  - CONTROL
  - SENIOR OPERATORS

  OPERATOR TRAINING REQUIRES 1 – 1.5 YEARS

FUEL REFABRICATION

- LWR – U OXIDE, MOX PELLETS; ZIRCALOY CLADDING; ZIRCALOY HARDWARE
- FBR – OXIDE, CARBIDE, OR NITRIDE PELLETS; SS CLADDING
- HTGR- CARBIDE, OXYCARBIDE, OXIDE KERNELS; TRISO MICROSPHERES; GRAPHITE MATRIX
  - FUEL FORM
    1. GRAPHITE SPHERES
    2. GRAPHITE PRISMS

LWR MOX FUEL REFABRICATION, te/yr

	2000	2005
• Belgium & France	175	195
• Japan	10	100
• Russia	60	
• UK	120	120
TOTAL:	**305**	**475**

REGULATORY CONNECTION

John H. Flack,
ACRS/ACNW

REGULATORY FRAMEWORK

- Licensing Process – Part 50, 52, 70
- Radiation Protection – Part 20
- Environmental Protection – Part 51
- Fuel Fabrication – Part 70
- HLW Vitrification and storage – Part 70
- Reprocessed uranium storage – Part 70
- Spent nuclear fuel storage – Part 72
- Cs/Sr and TRU storage – Part 30 and Part 70
- Transportation – Part 71
- Decommissioning – Part 50 and Part 51

REGULATORY AREAS

- NRC LICENSING OF FUEL RECYCLE FACILITIES
  - Safety & Security
  - Effluents to the environment
  - Wastes to disposal
- NRC OVERSIGHT OF OPERATION & ALARA
- DECOMMISSIONING

ENVIRONMENTAL PROTECTION REGULATIONS 10 CFR PART 51

- ENVIRONMENTAL REPORT
  - SUBMITTED BY APPLICANT
  - COMPLIANCE WITH TABLE S-3
- ENVIRONMENTAL ASSESSMENT
  - PERFORMED BY THE NRC
- ENVIRONMENTAL IMPACT STATEMENT
  - WRITTEN BY THE NRC

REPROCESSING PLANT ENVIRONMENTAL IMPACT STATEMENT

NECESSARY FOR SITING, CONSTRUCTION AND OPERATION

NO CURRENT EIS REGULATIONS SPECIFICALLY FOR REPROCESSING PLANTS

GESMO*
(AUGUST 1976)

- PURPOSE: BASIS FOR WIDE-SCALE USE OF MOX FUEL
- ASSUMPTIONS: 500,000 MWE LWR CAPACITY BY CY 2000
- ALTERNATIVES:
  - U + PU RECYCLE
  - U RECYCLE
  - NO RECYCLE
- KEY ENVIRONMENTAL FACTORS INCLUDED:
  - PLANT EFFLUENTS
  - PLANT WASTE GENERATED (CUBIC METERS)
  - OCCUPATIONAL DOSE
  - NON-OCCUPATIONAL DOSE

*GENERIC ENVIRONMENTAL STATEMENT ON THE USE OF RECYCLE PLUTONIUM IN MIXED OXIDE FUEL IN LIGHT WATER COOLED REACTORS

GESMO*
(AUGUST 1976)

- INCREASE IN PLANT EFFLUENTS (CURIES) FOR THE RECYCLE ALTERNATIVE OVER NO RECYCLE:
  - TRITIUM (APPROXIMATELY 2 ORDERS OF MAGNITUDE INCREASE)
  - CARBON-14 (ABOUT A FACTOR OF 3 INCREASE)
  - KR-85 (APPROXIMATELY 3 ORDERS OF MAGNITUDE INCREASE)
- INCREASE IN NON-OCCUPATIONAL DOSE (QUADRUPLED FOR FOREIGN POPULATION)
- DECREASE IN OCCUPATIONAL DOSE
- OVERALL CONCLUSION: NO CLEAR PREFERENCE FOR A SPECIFIED FUEL CYCLE OPTION ON THE BASIS OF WASTE MANAGEMENT CONSIDERATIONS.

EPA STANDARD

- 40 CFR PART 190 SUBPART B, "ENVIRONMENTAL STANDARDS FOR THE URANIUM FUEL CYCLE - 01/13/1977
- SPECIFIES ACCEPTABLE LEVELS OF RELEASES FOR OPERATION OF THE URANIUM FUEL CYCLE
- MAJOR ISSUES RAISED DURING REVIEW:
  - STANDARDS WOULD BE UNNECESSARILY CONSERVATIVE
  - DISAGREEMENT OVER NEED TO CONTROL Kr-85 RELEASES
  - DISAGREEMENT OVER RELATIONSHIP BETWEEN HEALTH EFFECTS AND DOSE

EPA STANDARD (CONTINUED)

- **BELIEVED THE INSTALLATION OF EFFLUENT CONTROLS WERE JUSTIFIED BY THE PUBLIC HEALTH BENEFITS ACHIEVABLE.**

Total quantity of release (per 1000 Mwe/yr):

- Kr-85 < 50,000 curies
  I-129 < 5 millicuries
  Pu + other < 0.5 millicuries

Maximum dose to any member of the public:

- Whole body ≤ 25 mrem
- Thyroid ≤ 75 mrem
- Any other organ ≤ 25 mrem

10 CFR Part 50 App. I
(ALARA for LWR Effluents)

- Dose values based on 40CFR190, to any individual:
- Liquid effluents ≤ 3 mrem whole body
  ≤ 10 mrem to any organ
- Gaseous effluents ≤ 5 mrem whole body
  ≤15 mrem to the skin
- Radioactive iodine & material ≤ 15 mrem to any organ

OPTIONS FOR LICENSING REPROCESSING FACILITY

- MODIFY CURRENT REGULATIONS
- NEW RULE (10 CFR PART XX)
- TECHNOLOGY-NEUTRAL FRAMEWORK (PART 53)

CURRENT LICENSING PROCESS - REPROCESSING FACILITY

- 10 CFR PART 50 – DOMESTIC LICENSING OF PRODUCTION AND UTILIZATION FACILITIES
- 10 CFR PART 52 – EARLY SITE PERMITS, DESIGN CERTIFICATION, COMBINED OPERATING LICENSE
- 10 CFR PART 70 – DOMESTIC LICENSING OF SPECIAL NUCLEAR MATERIALS

OPTION: MODIFY CURRENT REGULATIONS

- 10 CFR PART 50
  - PRO: EXPERIENCE WITH LICENSING STRUCTURE
  - CON: PRIMARILY USED FOR LICENSING LWRS
- 10 CFR PART 52
  - PRO: RISK-INFORMED, ONE STEP LICENSING PROCESS
  - CON: UTILIZES PART 50
- 10 CFR PART 70
  - PRO: EXPERIENCE WITH FUEL CYCLE FACILITY
  - CON: REQUIRES SUBSTANTIAL REVISION, CHANGE IN PHILOSOPHY

OTHER OPTIONS

- **NEW RULE**
  - **PRO: SPECIFIC TO REPROCESSING**
  - **CON: RESOURCE INTENSIVE, TIME**

- **NEW FRAMEWORK (10 CFR PART 53)**
  - **PRO: ONGOING INITIATIVE**
  - **CON: REACTORS ONLY**

ACNW LETTER (01/14/2002)
ISA VS PRA

- CHALLENGED THE NRC STAFF ON THE DECISION TO DEVELOP THE ISA METHOD TO RISK-INFORM NMSS ACTIVITIES, RATHER THAN EMPLOY PRA METHODS DIRECTLY

- QUESTIONED THE EFFECTIVENESS OF ISA IN LEADING TO DESIRED OUTCOMES

ACNW LETTER (CONTINUED)

RECOMMENDATIONS:

- MOVE TO QUANTITATIVE RISK ASSESSMENTS
- EXPLICITLY TREAT DEPENDENT FAILURES
- STRESS THE IMPORTANCE OF AGGREGATED RISK
- ENCOURAGE LICENSEES TO ACCOUNT FOR UNCERTAINTIES

10 CFR Part 50 App. F

(Policy Relating to Siting of Fuel Reprocessing Plants and Related Waste Management Facilities)

Requirements:

- High-level liquid waste limited to that produced in prior 5 years,
- Solidification of high-level liquid radioactive wastes and transfer of waste to a federal repository within 10 years following separation,
- High level waste disposed on land owned and controlled by the Federal government,
- Design objective must facilitate decommissioning,
- Provide information to ensure financial qualification.

KEY REGULATORY AREAS

- LICENSING APPROACH AND REGULATORY OVERSIGHT (Safety goals?)
- INTERGRATION OF EPA STANDARDS INTO NRC REGULATIONS (Dose criteria?)
- DESIGN CRITERIA FOR DECOMMISSIONING (Guidance?)

SUGGESTED ISSUES FOR ACNW CONSIDERATION

- **TECHNICAL**
  - MANAGING ^{129}I, ^{85}Kr, $^{14}CO_2$, ^{3}H
    1. APPROPRIATE MEASURES OF RISK
    2. TREATMENT TECHNOLOGIES
       1. STABILIZATION OF NOBLE GASES
       2. CLASSIFICATION AND DISPOSITION OF ^{129}I, $^{14}CO_2$
  - DISPOSITION OF SEPARATED ^{137}Cs AND ^{90}Sr (~30-YR $t_{1/2}$)
    1. INTERIM STORAGE FOR ~ 300 YRS
  - RECYCLE/DISPOSAL OF URANIUM

SUGGESTED ACNW ISSUES-2

- TECHNICAL (CONT.)
  - DISPOSAL OF LARGE VOLUMES OF GTCC (TRU) WASTE
  - MANAGEMENT OF UNCONVENTIONAL PYROPROCESSING WASTES
    - SODALITE HLW
    - FUSED HALIDE SALTS
  - ISSUES RELATED TO SAFEGUARDS(?)

SUGGESTED ACNW ISSUES-3

- REGULATORY
  - USE OF EXISTING REGULATIONS FOR LICENSING
    - WHICH ONES COULD BE USED?
    - WHAT CHANGES WOULD BE NEEDED?
  - NEW REGULATIONS
    - GENERIC OR SPECIFIC TO RECYCLE FACILITIES
    - TO WHAT EXTENT DETERMINISTIC AND HOW MUCH RISK-INFORMED?
  - IMPACTS ON OTHER REGULATIONS
    - CIVILIAN WASTE CLASSIFICATION SYSTEM
    - DOE EIS ON GTCC DISPOSAL

SUGGESTED ACNW ISSUES-4

- NEW REGULATIONS (CONT.)
  - ISSUES RELATED TO DECOMISSIONING
  - REGULATING RADIOACTIVE EFFLUENT RELEASES
  - BALANCING RISK, COST, AND TECHNOLOGY LIMITATIONS
    - » EPA ROLE: EXISTING 40CFR190 AND POSSIBLE FOLLOW-ON ACTIONS
    - » DOE ROLE: OUTCOMES OF RENEWED GEIS (SON OF GESMO)
    - » NRC ROLE: EXISTING 10CFR51 NOT CURRENTLY INTENDED FOR FUEL RECYCLE; NOTHING ELSE ON THE BOOKS

BACKUP SLIDES

ACNW MEETINGS IN FY2006

- HISTORICAL PERSPECTIVE ON U.S. NUCLEAR FUEL RECYCLE,
  06/07/2006- DR. R.G. WYMER

- NRC REGULATIONS FOR RECYCLING,
  06/07/2006 – NMSS/FCSS STAFF

- DOE/ANL - ADVANCED SEPARATIONS TECHNOLOGY DEVELOPMENT, 07/20/2006 – DR. J. LAIDLER

- DOE/INL – FUEL DEVELOPMENT AND ADVANCED FUEL RECYCLE FACILITY, 07/20/2006 – KEMAL PASAMEHMETOGLU

MOX FUEL FABRICATION STEPS

- URANIUM SOLUTION IS DENITRATED IN A FLUIDIZED BED TO FORM UO_2
- U AND Pu SOLUTIONS ARE MIXED, CONCENTRATED, AND CO-DENITRATED (BY MICROWAVE HEATING) TO PRODUCE A MIXED U/Pu OXIDE (MOX)

OXIDE (MOX) FUEL PREPARATION (CONT.)

- MIXED OXIDE IS CALCINED IN AIR IN AT 800 °C
- CALCINED PRODUCT IS HEATED IN A FURNACE IN H_2/N_2 AT 800 °C TO PRODUCE MOX FUEL MATERIAL
- (THIS TWO-STEP REDUCTION SAVES HYDROGEN)

COGEMA MOX FABRICATION FLOWSHEET

PELLET FABRICATION

UO_2 PuO_2 SCRAP

WEIGHING AND LOT PREPARATION

BALL MILLING

FORCED SIEVING

ADDITIVE MIXING

PRESSING W/HYDRAULIC PRESS

SINTERING

DRY CENTERLESS GRINDING

TESTING AND SORTING OUT

ROD FABRICATION

PELLET COLUMN PREPARATION

ROD FILLING

UPPER END PLUG TIG

ROD DECONTAMINATION

PRESSURIZATION, VENT-HOLE TIG SEALING

FINAL N/D TESTING

PACKAGING

STORAGE

TRANSPORTATION TO ASSEMBLY

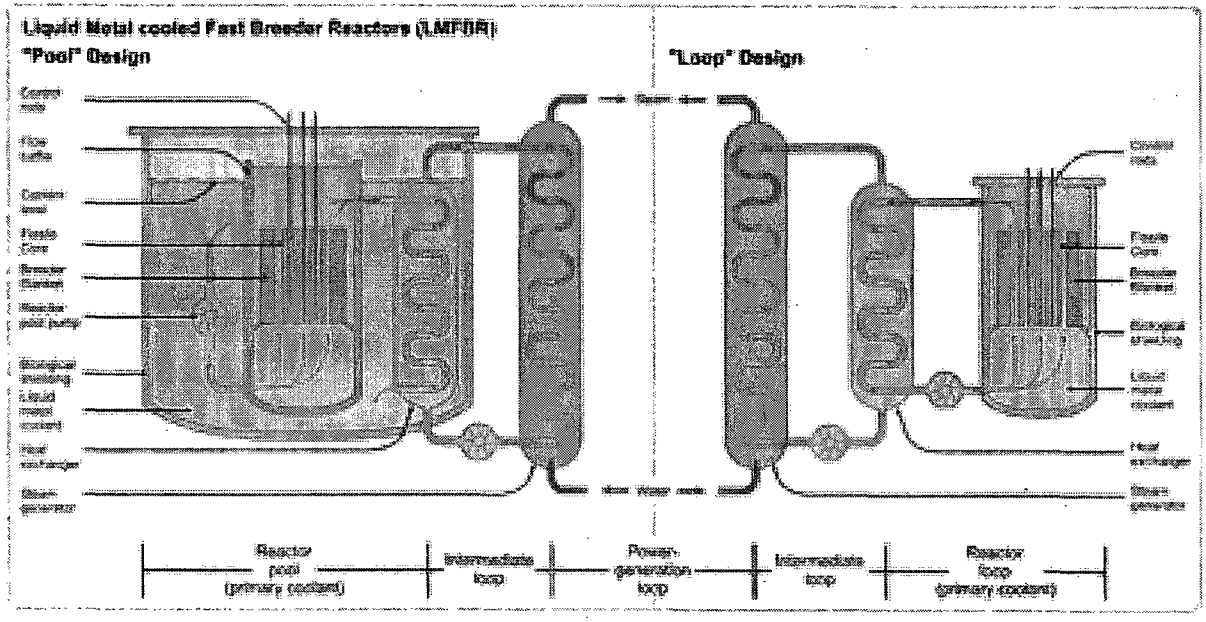
PRE-ASSEMBLY AND ASSEMBLING

FAST BREEDER REACTORS (FBRs)

TWO BASIC DESIGN TYPES:

- LOOP – HEAT EXCHANGERS EXTERNAL TO REACTOR TANK
- POOL – PRIMARY HEAT EXCHANGERS AND CIRCULATORS IN REACTOR TANK

DIAGRAM OF POOL AND LOOP FBRs



HTGR TYPES

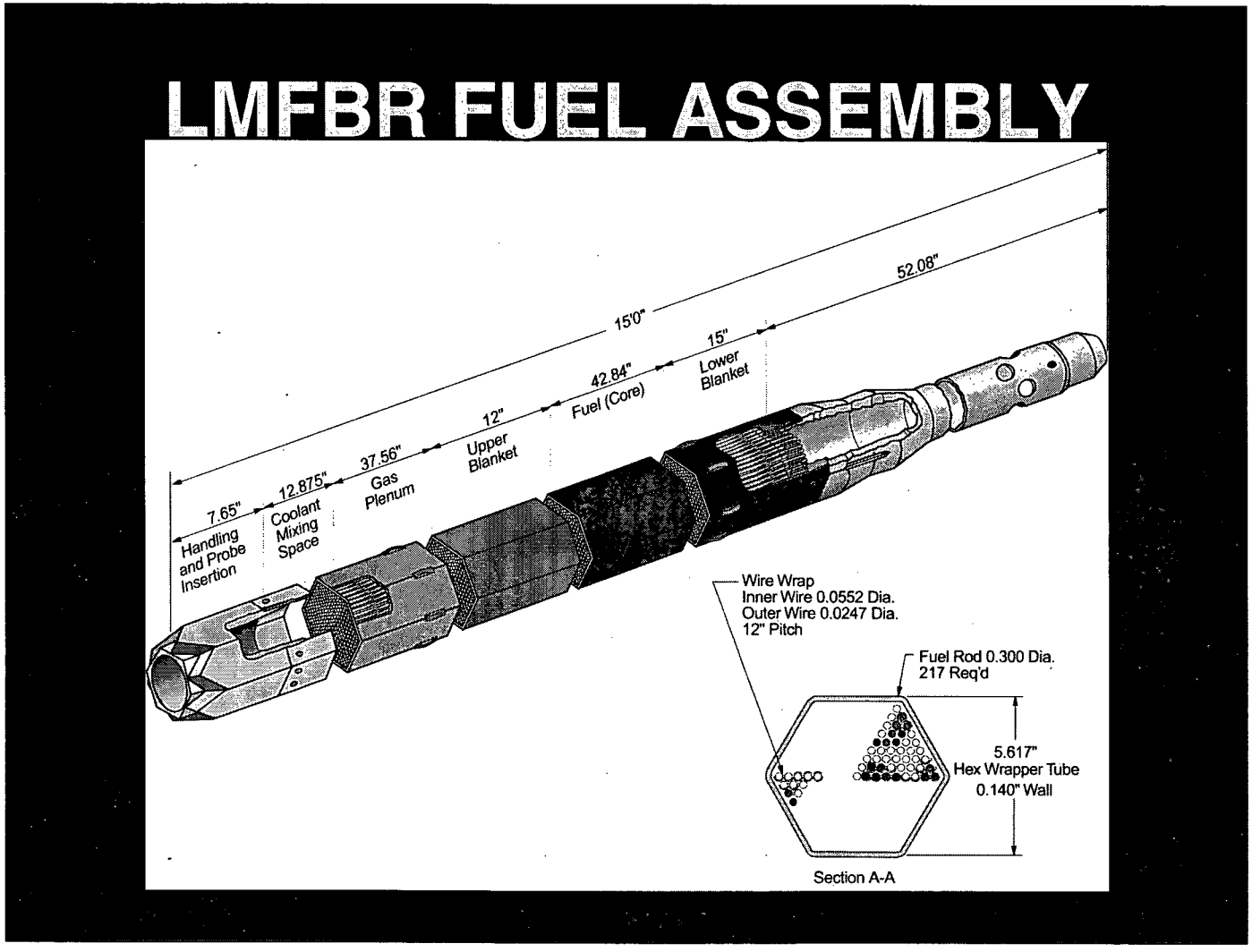
PEBBLE BED FUEL REACTORS

- DEVELOPED AND OPERATED IN GERMANY

PRISMATIC FUEL REACTORS

- DEVELOPED AND OPERATED IN U.S.

LIQUID METAL FAST BREEDER REACTOR FUEL



REACTOR FUELS

PWR

BWR

LMFBR

PRISMATIC

PEBBLE BED

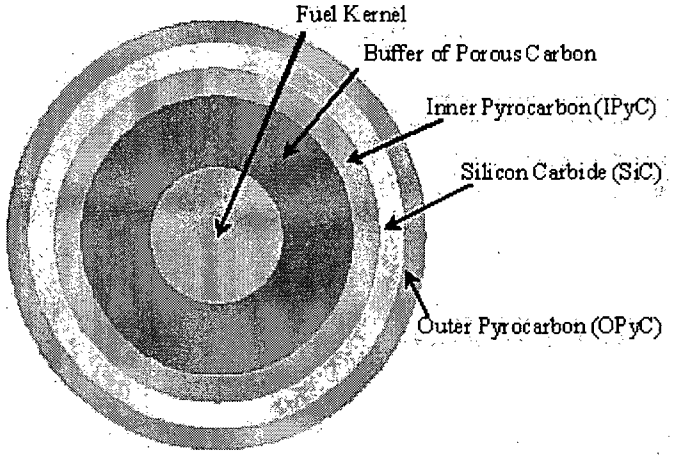
MOLTEN SALT

HTGR FUELS

TOP

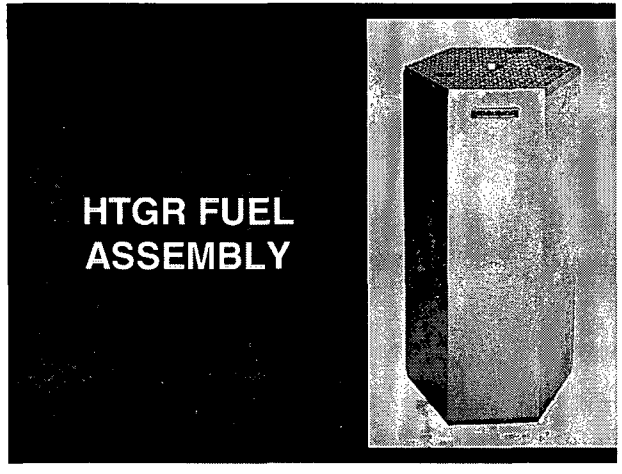
FUEL MICROSPHERE

CARBIDE OR OXIDE FUEL KERNEL

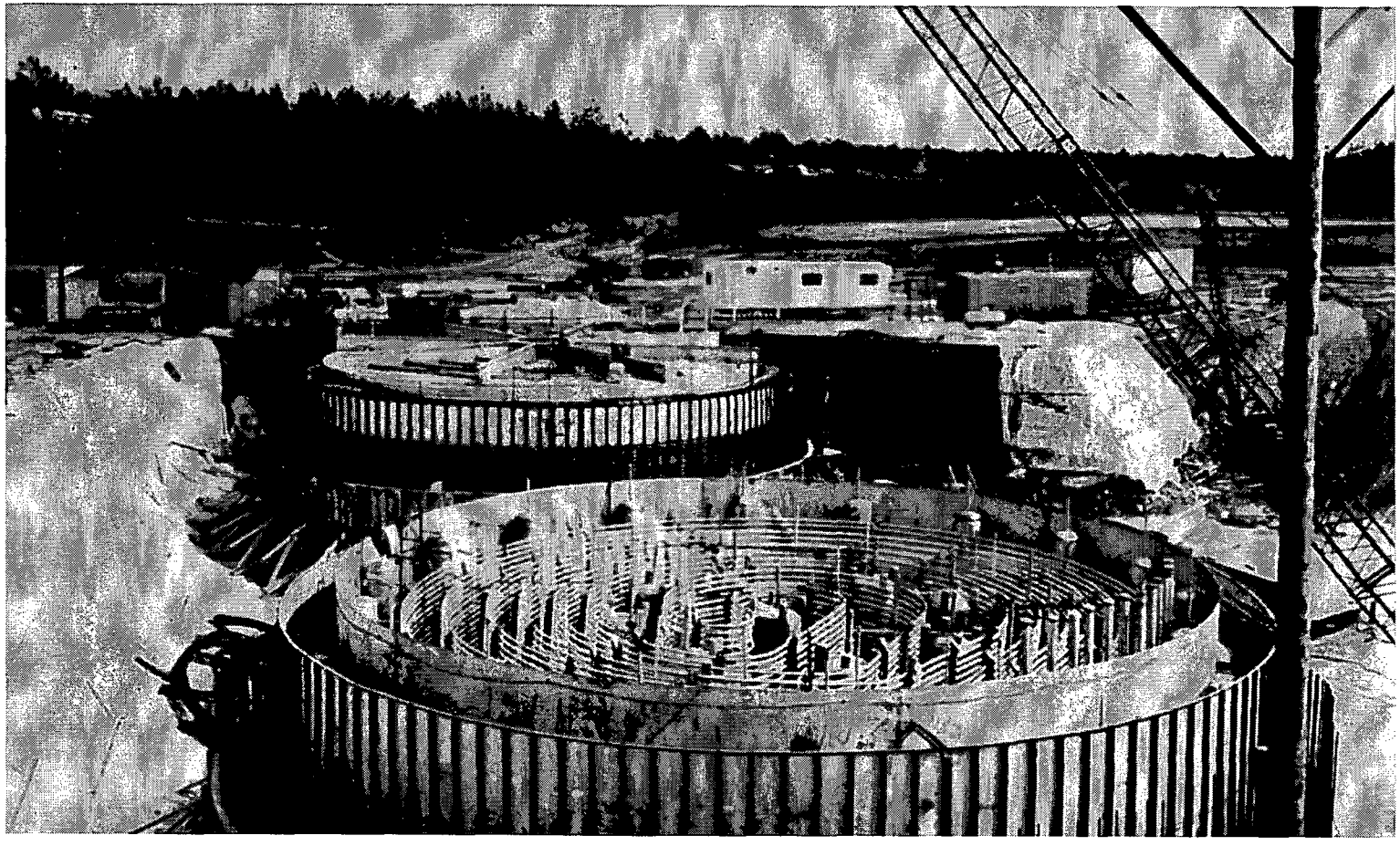


BOTTOM

PRISMATIC FUEL ELEMENT



AGNS HLW TANK UNDER CONSTRUCTION

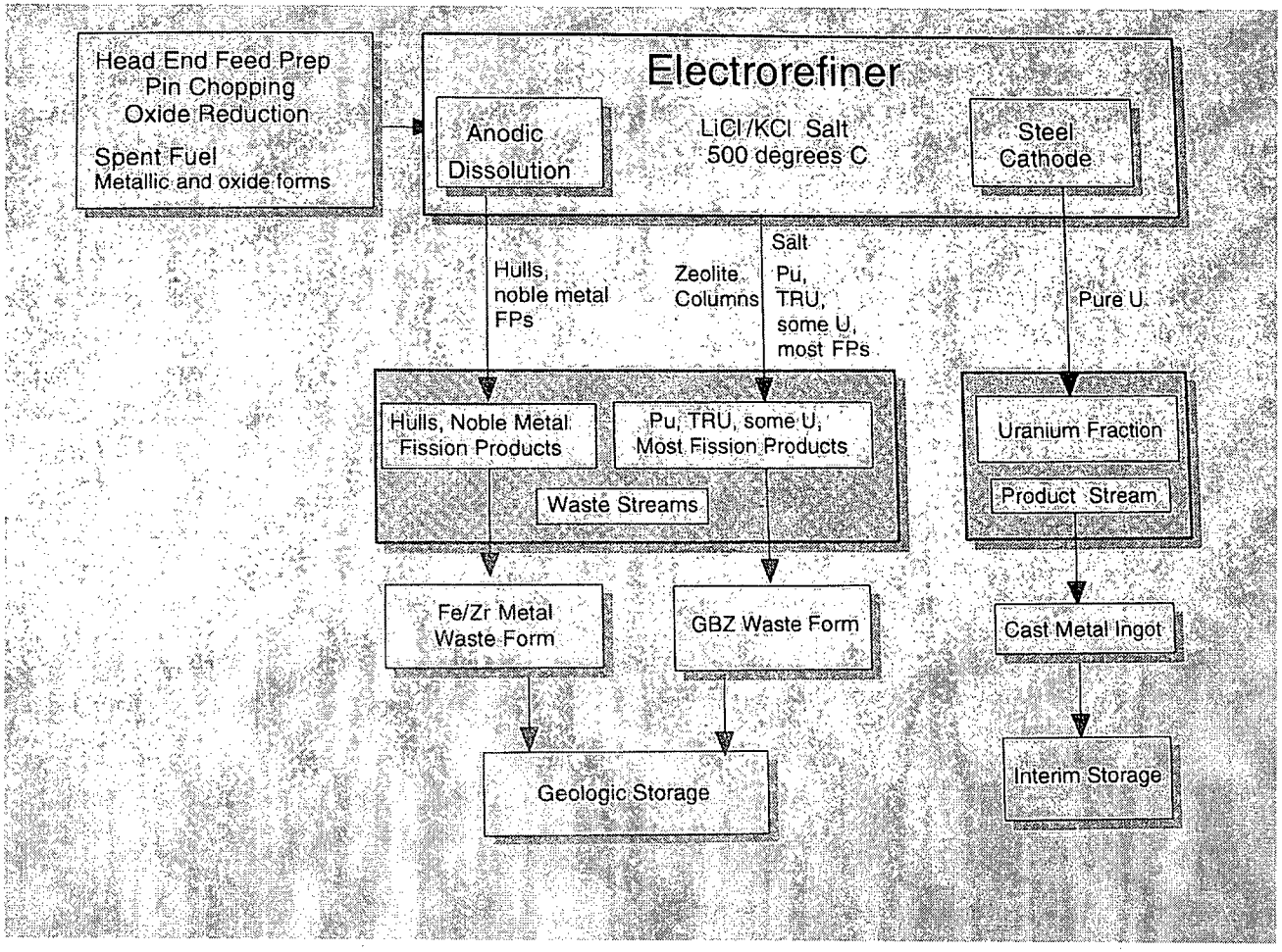


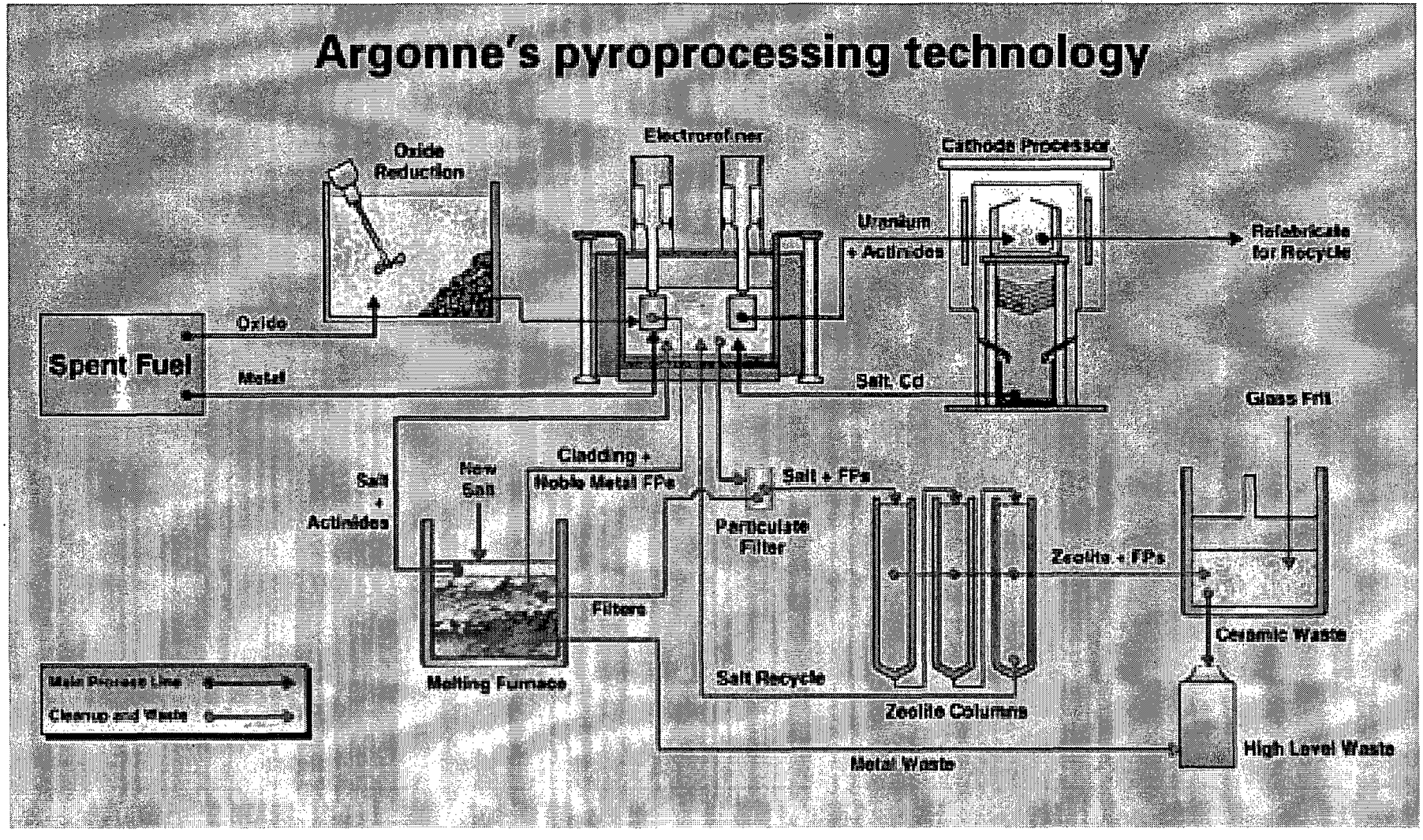
SURFACE OF SRP TANKS AFTER COMPLETION

HTGR FUEL REPROCESSING

- REMOVE EXCESS GRAPHITE
  - CRUSH AND/OR BURN
- BREAK SiC COAT
- ACID-LEACH FUEL KERNEL
- PUREX-TYPE SOLVENT EXTRACTION
- LAW GRAPHITE TO DISPOSAL
- OFF-GAS TREATMENT
  - ^{85}Kr, $^{14}CO_2$, ^{129}I

PYROPROCESSING




Argonne's pyroprocessing technology
Oxide Reduction
Electrorefiner
Cathode Processor
Uranium + Actinides
Refabricate for Recycle
Oxide
Spent Fuel
Metal
Salt Cd
Glass Frit
Cladding + Noble Metal FPs
Salt + Actinides
New Salt
Salt + FPs
Particulate Filter
Zeolite + FPs
Filters
Ceramic Waste
Main Process Line
Cleanup and Waste
Melting Furnace
Salt Recycle
Zeolite Columns
High Level Waste
Metal Waste