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AUSTRALIAN INSTITUTE OF NUCLEAR SCIENCE AND ENGINEERING

INIS-mf--11448

11th AINSE RADIATION BIOLOGY CONFERENCE

24th-25th AUGUST 1987

LUCAS HEIGHTS - AINSE THEATRE

CONFERENCE HANDBOOK

(Programme, Abstracts and General Information)

ISSN 0810 0497

AUSTRALIAN INSTITUTE OF NUCLEAR SCIENCE AND ENGINEERING

11TH AINSE RADIATION BIOLOGY CONFERENCE, 1987

LUCAS HEIGHTS, N.S.W.

Monday, 24th August, 1987	Commencing 10.30 a.m.
	Concluding 6.00 p.m.
	Conference Dinner 6.15 p.m.
Tuesday, 25th August, 1987	Commencing 9.00 a.m.
	Concluding 4.20 p.m.

Conference President

Professor P.A. Parsons	La Trobe University

Conference Committee

Professor P.A. Parsons	La Trobe University
Mr. J.K. Brown	ANSTO
Mr. D.R. Davy	ANSTO
Dr. D.G. MacPhee	La Trobe University
Assoc. Professor A.A. Morley	Flinders University of S.A.
Dr. M.F. Lavin	University of Queensland
Mr. E.A. Palmer	AINSE

Conference Secretary

Mrs. J. Watson	AINSE

CONTENTS

SUMMARY

Monday, 24th August, 1987 - Lucas Heights

10.30 - 10.40 a.m.	Opening Remarks Prof. P.A. Parsons - Conference President
SESSION I 10.40 - 12.30 p.m.	Mutagenesis Chairman: Dr. R. Brock (CSIRO)
12.30 - 1.30 p.m.	Conference Lunch - Stevens Hall Lounge & Dining Room
SESSION II 1.30 - 3.00 p.m.	Symposium: Food Irradiation Co-Chairmen: Mr. D. Davy (ANSTO) Dr. D.G. MacPhee (La Trobe Univ.)
3.00 - 3.20 p.m.	Afternoon Tea
3.20 - 4.40 p.m.	
SESSION III	Poster Session
4.40 - 6.00 p.m.	Stevens Hall Lounge & Dining Room (Includes pre-dinner drinks)
6.15 p.m.	Conference Dinner - Bamboo Room, ANSTO Canteen
7.45 p.m.	Conference Address - Prof. Sir Ernest Titterton (A.N.U.)

Tuesday, 25th August, 1987

SESSION IV 9.00 - 10.20 a.m.	Mammalian Radiation Biology I Chairman: Prof. A.A. Morley (Flinders Univ.)
10.20 - 10.40 a.m.	Morning Tea
SESSION V 10.40 - 12.40 p.m.	Mammalian Radiation Biology II Chairman: Dr. R. Martin (Cancer Institute)
12.40 - 1.40 p.m.	Lunch
SESSION VI 1.40 - 3.10 p.m.	Biological & Physical Dosimetry Chairman: Dr. M. Tyler (Univ. of Adelaide)
3.10 - 3.30 p.m.	Afternoon Tea
SESSION VII 3.30 - 4.20 p.m.	Radiation Responses - Nuclear Medicine Chairman: Dr. B.T.O. Lee (Univ. of Melbourne)

CONFERENCE SESSIONS

MONDAY, 24TH AUGUST, 1987 - LUCAS HEIGHTS

TIME	PAPER NO	
10.30 - 10.40		Opening Remarks - Conference President Prof. P.A. Parsons (La Trobe Univ.)
SESSION I		MUTAGENESIS Chairman: Dr. R. Brock (CSIRO)
10.40 - 11.10	1R Review	Mutagenesis in Procaryotes - Recent Developments. D.G. MacPhee (La Trobe Univ.)
11.10 - 11.30	2	Effects of Mutagenic and Antimutagenic Treatments on Precise Excision of Tn10. L.M. Hafner, D.G. MacPhee (La Trobe Univ.)
11.30 - 11.50	3	Frameshift Mutagenesis by Chloroquin in Salmonella Typhimurium and Escherichia Coli. D.G. MacPhee, K.A. Silburn (La Trobe Univ.)
11.50 - 12.10	4	Co-Mutagenicity of Commonly-Used Laboratory Glassware. D.M. Podger (CSIRO)
12.10 - 12.30	5	DNA Damage by Auger Electrons. R.F. Martin, G. D'Cunha, M. Pardee, V. Murray B.J. Allen (Peter MacCallum Cancer Inst. & ANSTO)
12.30 - 1.30		CONFERENCE LUNCH - Stevens Hall Lounge & Dining Room
SESSION II		SYMPOSIUM: FOOD IRRADIATION Co-Chairmen: Mr. D. Davy (ANSTO) Dr. D.G. MacPhee (La Trobe Univ.)
1.30 - 1.45	6R Review	Radiation Chemistry's Contribution to the Food Irradiation Debate. G.S. Laurence (Univ. of Adelaide)
1.45 - 2.00	7R Review	Ionising Radiation M.F. Lavin (Univ. of Queensland)
2.00 - 2.30	8R Review	Food Irradiation - The Consumer Perspective S. Heilpern (Australian Consumers' Assoc.)
2.30 - 3.00	9R Review	Status of Current Research Activities in Food Irradiation. P.A. Wills (ANSTO)
3.00 - 3.20		AFTERNOON TEA

MONDAY, 24TH AUGUST, 1987 (cont'd)

TIME	PAPER NO	
3.20 - 3.50	10R Review	Prospects for Food Irradiation in New Zealand. P.B. Roberts (D.S.I.R., N.Z.)
3.50 - 4.20	11R Review	A Review of Radiation Safety at a Gamma Sterilization Plant. F.P.J. Robotham (Univ. of Melbourne)
4.20 - 4.40		Discussion
SESSION III		POSTER SESSION - Stevens Hall Lounge & Dining Room
4.40 - 6.00	12	Comparison of γ-ray and Neutron Induced Mutations in DNA. S.P. Singh, M.F. Lavin, D. Cohen, N. Dytlewski, J. Boldeman (Univ. of Qld & ANSTO)
4.40 - 6.00	13	The Metabolism of ^{226}Ra and Ca by Freshwater Mussels and Turtles from Magela Creek. R.A. Jeffree (ANSTO)
4.40 - 6.00	14	Radiation and Feeding Effects on O_2 Uptake in Frog Tadpoles. H.C. Panter, J.E. Chapman, A.R. Williams (ANSTO)
4.40 - 6.00	15	Neutron Capture Therapy. B.J. Allen (ANSTO)
4.40 - 6.00	16	Ionising Radiation and its Application to Biotechnology. J.H. Williams, D.G. MacPhee (La Trobe Univ.)
4.40 - 6.00	17	Thymidine Catabolism and Ionising Radiation. T. Shaw, D.G. MacPhee (La Trobe Univ.)
4.40 - 6.00	18	The Effects of Adenosine 5'-Phosphates on 9-Aminoacridine-Induced Mutagenesis in Salmonella Typhimurium. G. Kopsidas, D.G. MacPhee (La Trobe Univ.)
4.40 - 6.00	19	Thymidine Catabolism as an Index of Toxicity. T. Shaw, R.H. Smillie, D.G. MacPhee (La Trobe Univ.)
4.40 - 6.00	20	Effects of Ionising Radiation on Aquatic Microenvironments. T. Shaw, R.H. Smillie, D.G. MacPhee (La Trobe Univ. & Univ. of Melbourne)
4.40 - 6.00	21	Morphological Changes in Human Melanoma Cells Following Neutron Capture Therapy. D.H. Barkla, B. Allen, et al (Monash Univ. & ANSTO))
4.40 - 6.00	22	Maternal Immune Response to Irradiation of Mouse Embryos. E.M. Nicholls (Univ. of N.S.W.)
6.15		CONFERENCE DINNER - Bamboo Room, ANSTO Canteen
7.45		CONFERENCE ADDRESS - Prof. Sir Ernest Titterton (A.N.U.)

TUESDAY, 25TH AUGUST, 1987

TIME	PAPER NO	
SESSION IV		Mammalian Radiation Biology I Chairman: Prof. A.A. Morley (Flinders Univ.)
9.00 - 9.30	23R Review	Severe Mental Retardation Following Fetal Irradiation. J.K. Brown (ANSTO)
9.30 - 10.00	24R Review	Critical DNA Target Size Model of Ionizing Radiation-Induced Mammalian Cell Death. I.R. Radford, G.S. Hodgson, J. Matthews (Peter MacCallum Cancer Inst.)
10.00 - 10.20	25	Cell Death by Apoptosis Following X-Irradiation of Neonatal Tissues. G.C. Gobé, B.V. Harmon, et al (Univ. of Queensland)
10.20 - 10.40		MORNING TEA
SESSION V		Mammalian Radiation Biology II Chairman: Dr. R. Martin (Cancer Institute)
10.40 - 11.00	26	DNA Topoisomerases Type I & II in Ataxia-Telangiectasia. M.F. Lavin, S. Kumar, S. P. Singh et al (Univ. of Queensland)
11.00 - 11.20	27	G2 Phase Delay in Ataxia-Telangiectasia and Control Cell Types. P.R. Bates, P. le Poidevin, M. F. Lavin (Univ. of Queensland)
11.20 - 11.40	28	Cellular Stress Induced by UV-Radiation and Hyperthermia - A Common Mechanism. K. Watson (Univ. of New England)
11.40 - 12.00	29	Evidence for Methylation Instructed Mismatch Repair in the Chromosomal DNA of CHO Cultured Cells. W. Diver, D. Woodcock (Peter MacCallum Cancer Inst.)
12.00 - 12.20	30	Isolation and Characterization of HLA-A2 Mutant Cells. M. Janatipour, K.J. Trainor, et al (Flinders Univ. of S. Australia)
12.20 - 12.40	31	Radiation Sensitivity of Human Natural Killer Cells Recognising Bone Marrow Targets. G. Flannery, E. Oliver (La Trobe Univ.)
12.40 - 1.40		LUNCH

TUESDAY, 24TH AUGUST, 1987 (cont'd)

TIME	PAPER NO	
SESSION VI		Biological & Physical Dosimetry Chairman: Dr. M. Tyler (Univ. of Adelaide)
1.40 - 2.10	32R Review	Micronuclei: An Important End-Point in Radiation Biology. M. Fenech (Flinders Univ. of S. Australia)
2.10 - 2.30	33	Outline of the Proposed Radioecology Study at the Maralinga Weapons Test Site. M.S. Giles (ANSTO)
2.30 - 2.50	34	In Vitro Radiosensitivity of Human Malignant Melanoma Cells to Low and High Let Radiation. J.K. Brown, M.M. Mountford et al (ANSTO)
2.50 - 3.10	35	Fast Neutron Irradiation Facilities at ANSTO - SNIF N.Dytlewski, D.D. Cohen et al (ANSTO)
3.10 - 3.30		AFTERNOON TEA
SESSION VII		Radiation Responses - Nuclear Medicine Chairman: Dr. B.T.O. Lee (Univ. of Melbourne)
3.30 - 3.50	36	Samarium - 153 Chelate Localization in Malignant Melanoma. J. Webb, P. Sorby, et al (Murdoch Univ.)
3.50 - 4.20	37R Review	Australian Radioisotopes Present and Future: A Presentation of the Australian Manufacture of Radio-Pharmaceuticals for Nuclear Medicine. K.W. Horlock (ANSTO)

ABSTRACTS

Mutagenesis in procaryotes - recent developments

D.G. MacPhee
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Bundoora, Victoria 3083.

Most induced mutagenesis results from the conversion of DNA damage or modifications into heritable changes in the nucleotide sequence. This implies that mutagenesis is a process which is intrinsic to the cell, operating in response to damage resulting from exposure to an extrinsic agent such as radiation or any one of many known chemical mutagens. It is likely that mutations are introduced into DNA at low frequwncy when enzymes such as polymerases and repair proteins encounter certain types of damage. Currently, many insights into mechanisms of mutagenesis are being obtained using both genetic and biochemical approaches. Knowledge is also accumulating suggesting relationships between the intial base sequence location in which particular lesions occur and the final location of mutational changes. On balance, the evidence at present tends to indicate that there may be only a few basic processes involved in generating mutations, although these same processes may be set in train by an enormously wide variety of agents. Whether or not induced mutations will occur (and at what frequency) can clearly by modulated by such factors as induction of repair systems, the action and types of DNA polymerases available, effects of the DNA sequence in the vicinity of the damage, and the presence or absence of antimutagenic agents.

EFFECTS OF MUTAGENIC AND ANTIMUTAGENIC TREATMENTS ON PRECISE EXCISION OF Tn10

J. M. Hafner & I.G. MacPhee

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Like many mutagens, transposable elements are capable of changing the genetic script, perhaps as a consequence of errors in the replication or repair of DNA. Transposable elements in prokaryotes include transposons (Tn elements) which can undergo both transposition and excision, the latter reaction being independent of transposon-promoted transposition and also of the normal host recombination genes such as the recA and recB,C genes. Amongst a variety of genetic rearrangements which bacterial transposons are capable of inducing, the simplest appears to be precise excision (the spontaneous and extremely accurate deletion of transposon nucleotide sequences). Understanding precise excision is important to a general understanding of how transposons are maintained and lost and hence how quite major changes to genetic sequences may occur. In addition, it appears that factors affecting precise excision may also be important to other DNA processes, including the formation of extended deletions and perhaps also general recombination. In an attempt to understand more about clean excision, we have used treatments which affect clean excision of Tn10, and involve both potentially mutagenic and anti-mutagenic activities. In this paper, attention will be focussed on possible effects of UV irradiation and anti-mutagens on clean excision of Tn10 in Salmonella typhimurium.

FRAMESHIFT MUTAGENESIS BY CHLOROQUIN IN SALMONELLA TYPHIMURIUM AND ESCHERICHIA COLI.

D.G. MacPhee and K.A. Silburn

Department of Microbiology
La Trobe University, Bundoora, Vic. 3083.

Chloroquine, commonly used as an antimalarial drug in South East Asia, Africa and other countries in which malaria is endemic, is deposited at high concentrations in mammalian tissues and its metabolites may remain in the body for up to five years. Reported results of mutagenicity tests with chloroquine have been either contradictory or would appear to have involved quite unacceptable protocols. We have found that chloroquine can be detected as a mutagen with the standard Ames tested strain TA97, but that its mutagenicity can be much more clearly demonstrated with an E. coli strain carrying the frameshift lacZ19124 marker. In addition, we have found that the mutagenicity of chloroquine is enhanced when aroclor-induced rat liver S9 is present, although this did not appear to be true for the highest dose-level tested (1,000 ug/plate). Reversion of the lacZ19124 marker by chloroquine was not significantly affected by the presence of plasmid pKM101, and recA, uvrC and umuC mutants were all about equally revertible by chloroquine. The dose response relationships obtained with all of the different strains are quite tightly clustered, and suggest that some (bacterial) system may have to be saturated before any mutagenic effects of chloroquine become apparent.

CO-MUTAGENICITY OF COMMONLY-USED LABORATORY GLASSWARE

by

D.M. Podger

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Abstract

9-aminoacridine (9AA) is a potent mutagen in strains of bacteria which detect frameshift mutations at -CCCC- sites. Unlike many other chemical mutagens which induce mutations indirectly as a result of SOS-Repair, little is known of the mechanism of frameshift mutagenesis at these sites.

In an investigation of chemicals which alter the yield of induced mutants in Salmonella typhimurium strain hisC3076 following treatment with 9AA, the vessel in which cells were incubated was often found to enhance the yield of mutants. A co-mutagenic effect was found in a range of disposable polypropylene plasticware including 1.5 ml microcentrifuge tubes from different sources, 50 ml centrifuge tube and sterile syringes from a number of sources. Plasticware made from polystyrene (petri dishes) and polyethylene was not found to be co-mutagenic. Incubations of cells in any vessel in the absence of 9AA was found to be non-mutagenic.

The reported potentiation of 9AA-induced frameshift mutagenesis by potassium chromate (1) is not a likely explanation as the manufacturing process for the most "active" plasticware does not involve chromate.

A similar potentiation phenomenon of enhanced yields of induced mutants on plates containing any one of naladixic acid, ethyl methanesulfonate, cinnamaldehyde or bleomycin (all of which are non-mutagenic in strain hisC3076 in addition to 9AA has been shown to be dependent on the uvrD gene function (2). The relationship between this type of potentiation and polypropylene co-mutagenesis is being tested using a uvrD derivative of hisC3076.

References

[1] J.M.LaVelle (1986) Mutation Res.,171,1-10
[2] D.M.Podger and G.W. Grigg (1986) Mutagenesis,1,283-286

DNA DAMAGE BY AUGER ELECTRONS

by

R. F. Martin, G. D'Cunha, M. Pardee, V. Murray and †R. J. Allen

Molecular Science Group,
Peter MacCallum Cancer Institute, Melbourne
and
†Lucas Heights Research Laboratories, ANSIO

Abstract

The low energy Auger and Coster-Kronig electrons that are emitted following ionisation, electron capture or internal conversion constitute a highly focussed source of radiochemical damage. For example, theoretical studies of ^{125}I indicate that an average of more than twenty low energy electrons are emitted per decay event, and energy deposition in the vicinity of the decaying atom is more intense than that from the passage of a 5 MeV α-particle [1]. Decay of an ^{125}I atom covalently bound to DNA induces single strand breaks in both strands, the majority of breaks being within 3-4 base pairs of the decaying atom [2], so a double-strand (ds) break is a frequent outcome. Experiments with ^{125}I-labelled DNA ligands have shown that a ds DNA break also results from decay of ^{125}I that is non-covalently associated with DNA [3]. In this paper we will report the extension of experiments with ^{125}I DNA ligands to studies with intact cells and also the results of experiments with ^{67}Ga-labelled DNA ligands which also induce ds DNA breaks. ^{67}Ga, like ^{125}I, decays by electron capture.

Finally we shall describe our recent exploration of a previously unrealised source of Auger electrons; the induction of ds DNA breaks following thermal neutron capture by DNA-bound ^{157}Gd atoms. The ^{157}Gd (n,γ) ^{158}Gd reaction is accompanied by internal conversion, and we presume that the associated Auger and Coster-Kronig electron emission results in the damage to DNA.

References

[1] Charlton, D.E. Radiat. Res., 107, 163-171 (1986).
[2] Martin, R.F. and Haseltine, W.A. Science, 213, 896-898 (1981).
[3] Martin, R.F. Int. J. Radiat. Biol., 32, 491-497 (1977).

RADIATION CHEMISTRY'S CONTRIBUTION TO THE FOOD IRRADIATION DEBATE

by

G.S. Laurence

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Abstract

Within less than a nanosecond of the initial absorption of ionizing radiation by foodstuff the physical processes are replaced by chemical reactions which are responsible for any subsequent changes in the chemical, physical, biological, and nutritional properties of the material. The proper understanding of the primary and secondary radiation induced chemical reactions provides the framework for an informed discussion of the effects of using ionizing radiation in food processing. The chemical changes produced by ionizing radiation are the result of normal free radical reactions which in the case of foods are comparable to those induced by other processes, particularly those involving high temperatures. The radiation chemistry of food components does not indicate any special sensitivity of minor constituents such as vitamins.

Despite the chemical and physical complexity of most foods, the radiation chemistry data may be used to evaluate reported results from the irradiation of actual foods, to establish the nature and importance of radiolytic products, and to contribute to the establishment of regulatory processes and the analytical procedures which would be needed. Radiation chemistry also provides an insight into many of the questions raised by those who fear the use of radiation processing to permit the consumption of food which would otherwise be rejected by the consumer.

IONISING RADIATION

by

M.F. Lavin

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Brisbane, Australia

Abstract

Ionising radiation is cytotoxic, mutagenic and carcinogenic. Its utilisation in food irradiation is clearly as a cytotoxic agent to prevent contamination and spoilage. The damaging effects of ionising radiation on cells are widespread and include both direct and indirect mechanisms. DNA is an important target for both forms of damage which cause base modification and breaks to the phosphodiester backbone of the molecules. Indirect damage to DNA is primarily due to hydroxyl radicals which arise from radiolysis of water. The hydroxyl radical is involved in addition reaction with individual bases as well as in abstraction of hydrogen atoms from deoxyribose, ultimately giving rise to breaks in DNA.

These breaks in the backbone of DNA and modified nucleotides can be repaired by a number of different mechanisms and involve removal of nucleotides and re-sealing of phosphodiester bonds. The overwhelming doses used in food irradiation saturate the system giving rise to non repairable lethal lesions.

FOOD IRRADIATION - THE CONSUMER PERSPECTIVE

by

Sandra Heilpern

Australian Consumers' Association

Abstract

ACA began an Inquiry into Food Irradiation in October 1986 and presented its Report to the Minister of Health in May 1987.

The submissions to the Inquiry included concerns about the effects of the toxicity and nutritional losses in irradiated foods.

The Inquiry found that there were low risks of toxicity and of significant vitamin losses, that the technology had both practical and economic limitations, and that it was not used widely on a global scale.

To accommodate the possibility of a potentially viable industry ACA recommended a legislative framework and set of controls to be put in place prior to the commercial development. These included national control under a Food Irradiation Act, of all aspects of the industry, item by item approval rather than blanket approval, and strict labelling regulations.

It was recommended to the Government that food irradiation go ahead conditional upon these recommendations being adopted.

STATUS OF CURRENT RESEARCH ACTIVITIES IN FOOD IRRADIATION

by

P. A. Wills

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Abstract

Over the last 40 years at least 50 countries have undertaken research into the process of using ionising radiation from high energy electrons, X-rays, or from the gamma radiation producing isotopic sources of ^{60}Co or ^{137}Cs to improve the storage life or hygienic quality of foods or to meet quarantine requirements. About a dozen countries have made the transition from research and development to commercial reality, albeit with generally a narrow range of foods and relatively low volumes being treated.

Research into food irradiation began in Australia in 1960, was largely deferred towards the end of the 1960s and became revitalised a decade later when the WHO accepted the Codex Alimentarius Commission's first international standard for irradiated foods. The chief potential applications now being investigated by collaborative projects between ANSTO and the Gosford Post-Harvest Horticultural Laboratories, Queensland Department of Primary Industry (QDPI) and the CSIRO Meat Research Laboratory are the uses of radiation as a disinfestation treatment for fruit intended for export and for extension of shelf-life of chilled meats and seafoods. The QDPI is also undertaking research into the detection of irradiated foods. In addition, foods irradiated in S.E. Asian countries are being evaluated after shipment to Australia under commercial conditions as part of the IAEA/RCA Asian and Pacific Regional Coordinated Research Project on Food Irradiation, Phase II. Some results of these investigations will be presented.

A vast amount of research data has been amassed world-wide to assess the wholesomeness of irradiated foods. The need for even more research is being called for by some concerned groups in Australia and other countries. The relevance of some of the areas of concern will be discussed.

PROSPECTS FOR FOOD IRRADIATION IN NEW ZEALAND

by

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Abstract

The New Zealand Food Regulations 1984 state that the sale of irradiated food is illegal without the specific consent of the Minister of Health. This permission is believed to have been sought twice. One batch of various herbs and spices was permitted to be decontaminated with a dose not to exceed 10 kGy in early 1985. The other application was refused on the grounds that insufficient need for the process was shown.

The Department of Health issued a position paper in 1986 stating that food irradiated in accordance with the Codex General Standard 106-1983 was safe. Other statements have made clear the intention to ban the use of the chemical fumigant EDB. This and the expectation that the use of chemical fumigants will come under increasing threat of international bans has raised the possibility of irradiation as one of the possible alternatives as a horticulture quarantine treatment.

The potential value of this use of radiation and the preliminary research that has been done will be discussed. Uses other than as a quarantine use will also be briefly mentioned.

The Ansell-Steritech application for permission to build a medical product sterilisation irradiator in Auckland has, therefore, come at a time when the irradiation issue was becoming more widely discussed. Its ability to treat food, if permitted, has catalysed public debate on food irradiation, with many of the objections to the plant being on the grounds of food irradiation potential.

The government has set up an Irradiation Issues Working Party. It comprises nine government departments, but is also charged with wide consultation with industry, environmental and consumer groups. A report to Cabinet is expected by April 1988.

A REVIEW OF RADIATION SAFETY AT A GAMMA STERILIZATION PLANT

by

F.P.J. Robotham

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Parkville, Australia

Abstract

Objections to the irradiation of food are based mainly on two broad premises:

i) that damage is done to the food by the irradiation process posing untoward hazards to the consumers of the food and;

ii) the use of large radiation sources produces unwarranted radiation exposure to both plant operators and members of the public.

This paper considers the second point and concludes that under normal operating conditions the risks from actual and potential radiation exposures are well below acceptable levels.

The author was chairman of a panel that reviewed, on behalf of the Victorian Health Department, radiological safety at a large (40 PBq) gamma sterilization plant. This paper is based on the panels report.

COMPARISON OF ϒ-RAY AND NEUTRON-INDUCED MUTATIONS IN DNA

by

S.P. Singh[1], M.F. Lavin[1], D. Cohen[2], N. Dytlewski[2], J. Boldeman[3]

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2 AINSE,
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3 ANSTO,
Lucas Heights, Sydney, Australia

Abstract

Exposure of DNA to ionizing radiation gives rise to a number of different lesions and a number of investigations on radiation-induced mutations showed no specificity as to the target bases (Shinoura et al., 1983; Kato et al., 1985). More recently Ayaki et al., (1986), using M13 mp 10 phage DNA damaged by ϒ-rays, have shown that the changes induced are largely base substitutions when the phage is tranfected into E. coli. The predominant base change was a cytosine to thymine transition.

Methods for sequencing DNA allow for the rapid detection of nucleotide changes to DNA after exposure to radiation. In this study we intend to investigate mutations induced in the lac Z gene in M13 after exposure to high and low LET radiation.

A ^{60}Co source (90 Gray/min) has been used to determine survival of M13 at doses up to 400 Gray. A system for delivering equally effective doses of high energy neutrons has been set up using the Van de Graaff accelerator at Lucas Heights.

THE METABOLISM OF ^{226}RA AND CA BY FRESHWATER MUSSELS AND TURTLES FROM MAGELA CREEK

by

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Abstract

Freshwater musels and turtles from Magela Creek, N.T., are components of the traditional diet of aboriginals living downstream of the Ranger Uranium Mine. A number of studies have been conducted to evaluate their ability to accumulate ^{226}Ra, when exposed to mine effluent. The paper draws on these findings to elucidate the biological and radioecological attributes of these lifeforms that explain their differential capacity to accumulate ^{226}Ra.

RADIATION AND FEEDING EFFECTS ON O_2 UPTAKE IN FROG TADPOLES

by

H.C. Panter, J.E. Chapman and A.R. Williams

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Abstract

The oxygen consumption of tadpoles of the frog Limnodynastes tasmaniensis was tested at 25°C under varying conditions of feeding or starvation and radiation.

The results revealed differences in the oxygen consumption of fed and starved non-irradiated tadpoles. However, fed irradiated tadpoles showed no significant changes after radiation up to 5 Gy, whereas the oxygen uptake of starved tadpoles increased even at a dose of 0.5 Gy compared with unirradiated starved controls.

Thus tadpoles already stressed by starvation appear to be the ones most likely to show an effect from the further stress of radiation.

NEUTRON CAPTURE THERAPY

by

B.J. Allen

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Abstract

The principles of neutron capture therapy for cancer are described. The Australian research program covers the many disciplinary facets involved in NCT, and an overview of the current research program is presented. Cancers being examined are malignant melanoma, leukaemia and glioblastoma, each of which offers specific advantages for in vitro and in vivo experiments in NCT.

IONISING RADIATION AND ITS APPLICATION TO BIOTECHNOLOGY

by

J. H. Williams and D. G. Macphee

Department of Microbiology
La Trobe University
Bundoora, Victoria

Abstract

With the advent of recombinant DNA technology it has become possible to cross species barriers and create micro-organisms that can produce a wide range of foreign proteins. Efficient expression of large amounts of these proteins has been the subject of much research. Plasmid amplification by chloramphenicol/spectinomycin treatment, cloning downstream of highly efficient promoters and the use of multicopy plasmids have all proven to be satisfactory ways of maximising foreign gene expression in *E. coli*.

In 1983 it was found that exposure of *recA* bacteria to γ-irradiation produced non-dividing cells which could sustain the multiplication of "small" plasmids. Yields of plasmid DNA obtained using this method compared favourably with those obtained by CsCl gradient centrifugation [1]. This method of increasing plasmid DNA yield is based on the observation that *recA* mutants cannot repair irradiation-induced damage to their chromosomal DNA and in fact the damaged chromosome is further degraded by the action of endogenous nucleases (the *recB,C* gene products). Owing to their size, small extra-chromosomal elements such as plasmids survive this treatment since they do not present a large enough target and therefore, in the majority of cases, are not damaged by the radiation. Thus, when a plasmid-containing *recA* mutant is exposed to radiation, the chromosomal material is destroyed while the plasmid remains intact. This being so, protein synthesis in these irradiated cells should be directed only by the plasmid and not by the chromosome, ie only the intact plasmid-encoded mRNA should continue to be translated into protein.

Results of experiments designed to test this idea will be presented.

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THYMIDINE CATABOLISM AND IONISING RADIATION

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Substantial evidence suggests that the most potentially lethal lesions produced by ionising radiation are single- and double-strand breaks in cellular DNA [1]. After exposure of cells to sublethal radiation, the majority of DNA lesions is rapidly repaired [2], but repair processes, which cope well with small amounts of radiation-induced DNA damage, fail to operate accurately or effectively if the damage is more severe [3].

Both high fidelity DNA repair and high fidelity DNA replication require balanced pools of deoxynucleoside triphosphate (dNTP) DNA precursors and imbalance in dNTP pools may cytostatic or even lethal [4]. Convincing arguements can be presented in support of the postulate that the intracellular availability of thymidine triphosphate (dTTP) is the ultimate regulator of intracellular dNTP pools and therefore of DNA synthesis. In cells which salvage thymidine, intracellular dTTP levels are determined by the balance between anabolic and catabolic pathways which compete for substrate. In dying cells the latter predominates.

Mammalian blood contains high levels of thymidine phosphorylase, a (normally) catabolic enzyme specific for 2'-deoxynucleosides [5,6]. Here we report the results of experiments in which peripheral venous blood from normal, healthy donors, was used to test whether ionising radiation affects thymidine catabolism. Our results show that blood thymidine phosphorylase activity is inhibited by exposure to low-dose ionising radiation, but stimulated by exposure to higher doses. We are currently investigating the molecular mechanism(s) responsible for this phenomenon, which appears to involve calcium dependent processe(s).

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The Effect of Adenosine 5'-Phosphates on 9-Aminoacridine-Induced Mutagenesis in *Salmonella typhimurium*

by

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Frameshift mutagenesis by 9-aminoacridine (9AA) involves intercalation of flat, planar 9AA molecules into DNA. Such intercalation causes damage (or perhaps distortion of the double helical structure of the DNA) which in turn appears to induce an error-prone DNA repair mechanism. The way in which this repair mechanism is controlled has been studied using strains of *Salmonella typhimurium* carrying the *hisC3076* frameshift marker.

Results presented in the poster will illustrate the effect of adenosine 5'-phosphates on the number of 9AA-induced His^+ revertants.

Cells grown in liquid culture exposed to 9AA (10µg/ml) show a high reversion rate to prototrophy. When either adenosine 5'-triphosphate (ATP), adenosine 5'-diphosphate (ADP), adenosine 5'-monophosphate (AMP), or adenosine 3'5'-cyclic monophosphate (cAMP) was added to the liquid culture, significantly fewer 9AA-induced His^+ revertants were recovered. These findings will be interpreted in the light of other evidence that 9AA-induced mutagenesis involves an inducible pathway which can be modulated by changing environmental conditions.

THYMIDINE CATABOLISM AS AN INDEX OF TOXICITY

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Most living cells are able to salvage nucleosides. The thymidine kinase pathway (thymidine -TMP-TDP-TPP) is often considered the most important salvage pathway because TTP is (1) uniquely a DNA precursor and (2) regulates the activity of several enzymes concerned with other salvage pathways or with DNA synthesis. The incorporation of labelled thymidine into DNA is the basis for many assays used to quantify cell division, immunocompetence and genotoxicity. In these assays the role of thymidine catabolism is frequently overlooked.

Both prokaryotic and eukaryotic cells contain cytoplasmic phosphorylases which degrade nucleosides to their respective bases. In most normally cycling cells, the activity of the degradative pathway appears to be inversely proportional to thymidine kinase activity. In any complex biological system, the measurement of thymidine degradation may therefore provide information complementary to that provided by measurements of incorporation of labelled thymidine into DNA, which relates only to S-phase cells. Furthermore, the measurement of induced perturbations of the catabolic pathway may provide information which complements and extends that obtained by conventional assays based on thymidine incorporation into DNA.

We have devised a simple assay based on HPLC for measuring the rate of thymidine catabolism under saturating conditions in a variety of natural ecosystems. By preincubating samples with different concentrations of known cell poisons and environmental contaminants before assay we have been able to demonstrate dose-response relationships which could previously be demonstrated only with difficulty, if at all. We anticipate that assay of thymidine catabolism may become the basis for a new and highly sensitive class of toxicology assays.

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EFFECTS OF IONISING RADIATION ON AQUATIC MICROENVIRONMENTS

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Previous workers have shown that one of the earliest consequences of exposure of cells to agents which damage chromosomal DNA is inhibition of DNA synthesis as measured by incorporation of labelled thymidine into newly synthesised DNA [X-X]. Inhibition of thymidine incorporation may be achieved by reduction of uptake, increased catabolism, blockage of DNA polymerase activity, or any combination of these possibilities.

We have recently developed rapid methods for measuring thymidine catabolism in a variety of complex natural ecosystems and have investigated the possibility that in these systems, catabolism of exogenously supplied thymidine may, under certain circumstances, be indicative of cytotoxicity and DNA damage.

Here we present preliminary results of experiments in which ionising radiation was used as a inducer of DNA damage. The experiments were designed to test whether dose-response relationships between thymidine catabolism and (presumptive) DNA damage could be demonstrated in complex biological systems provided that certain conditions were met. The results suggest that thymidine catabolism may be a useful index of cytotoxicity.

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MORPHOLOGICAL CHANGES IN HUMAN MELANOMA CELLS FOLLOWING NEUTRON CAPTURE THERAPY

by

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Malignant human melanoma cells from two established lines, MM96L and MM418,[1] were placed in flasks containing RPMI medium 1164 supplemented with 20% FCS, Gentomycin and Fungizone. After 48 hrs an enriched boron-10 analogue of phenylanaline[2] (10 $\mu g\ mL^{-1}$ BPA) was added to the medium. After 20 hrs the medium was replaced with non-BPA containing medium and one hour later these flasks of cells together with flasks of untreated control cells were placed in the neutron capture therapy facility of the Moata reactor and exposed to a neutron fluence of 10^{13} n cm^{-2}. The total radiation dose was 5.40 Gy, 30% of which is ascribed to gamma radiation. At time intervals of 4, 8, 18, 27, and 150 hrs later cells were fixed in 5% gluteraldehyde, dehydrated through alcohols, embedded in Epon and examined using light and electron microscopy.

During the first 18-27 hrs after treatment both cell lines continued to divide and cell damage was not evident. The two cell lines then responded differently. At 27 hrs MM96L cells began to detach from the flask and by 47 hrs showed ultrastructural signs of damage, initially in the nucleoli and later in the cytoplasm. Boron +ve and boron -ve cells responded similarly. There was no evidence of melanosome formation.

MM418 cells continued to divide up to 48 hrs after treatment and failed to show morphological evidence of cell damage. Instead, they differentiated into 4 or 5 morphologically distinct subtypes and melanasome production increased. Cell division ceased before confluence was reached and these cells remained alive 7 weeks after irradiation. Differences between boron +ve and -ve cells were not apparent.

The study shows that the response of human malignant melanoma cells to high LET radiation is cell-line dependent. Further studies using other doses of 10-BPA and other biologically active boron compounds will now be undertaken to clarify the observations reported in this preliminary study.

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MATERNAL IMMUNE RESPONSE TO IRRADIATION OF MOUSE EMBRYOS

by

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Abstract

As previously reported maternal lymphocytes were labelled with quinacrine and traced into the embryos, with and without γ-irradiation. More definitively, 11 day mouse embryos, irradiated and unirrradiated, were examined in smear preparations stained using the α-naphtyl butyrate esterase method (Sigma). Striking responses of the maternal immune system in the irradiated embryos were observed for the first time. The dose response and time course of these responses will be reported.

SEVERE MENTAL RETARDATION FOLLOWING FETAL IRRADIATION

by

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Abstract

During fetal development the neuroblasts in the primitive human brain are undergoing rapid multiplication prior to their differentiation into neurons when no further proliferation or replacement can occur. Neuroblasts are highly radiosensitive to the lethal action of ionising radiation. The late radiation-induced effects on the fetal brain are manifested as significantly reduced intelligence, small head size and/or severe mental retardation (SMR), of which the latter is the most serious effect.

Based on the frequency of occurrence of SMR in the Japanese atomic bomb survivors exposed in utero the period of maximum sensitivity occurs from 8-15 weeks post conception and shows a striking correlation with the greatest multiplication of neuroblasts and their migration to the cerebral cortex. The dose response relationship appears to be linear with no threshold with an increase in the frequency of SMR of 0.40 Gy^{-1}, e.g. at a dose of 0.01 Gy low-LET radiation about one case of SMR per hundred exposed individuals might be expected. (The natural incidence of SMR in Australia and European countries is about 0.8%).

The practical implications of radiation-induced SMR in the radiological protection of female members of the public and in occupationally exposed women known to be pregnant will be discussed.

CRITICAL DNA TARGET SIZE MODEL OF IONIZING RADIATION-INDUCED MAMMALIAN CELL DEATH

by

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ABSTRACT

A new model of mammalian cell killing by ionizing radiation will be discussed. This model, termed the critical DNA target size model, is based largely on recent experimental results from our laboratory. The model incorporates four basic hypotheses: (1) DNA double-strand breakage (dsb) is the critical radiation-induced lesion, (2) DNA dsb within critical regions (targets) of the genomic DNA produce lethal events and this can be expressed mathematically as $S = (1 - X_c/L)^N$ where S is survival, L is the genome length, X_c is the genome length occupied by critical targets, and N is total DNA dsb, (3) nuclear DNA continuity is prevented by chromosome fragment loss, and (4) cell death is caused by karyotypic discontinuity.

Experimental evidence contrary to other radiation action models will be examined and the hypotheses of our model will be justified.

CELL DEATH BY APOPTOSIS FOLLOWING X-IRRADIATION OF NEONATAL TISSUES

by

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Abstract

A morphological study was undertaken to determine the type of cell death induced by X-irradiation in the developing immature kidney, cerebellum and testis. Three to 5 day old male Sprague-Dawley rats were exposed to a whole body dose of either 2 or 5 Gy. Irradiated and control tissues were studied by light and electron microscopy, at various times ranging from 2 to 24 hours. Selected cells in all three tissues are known to be radiosensitive at this time of development: in the kidney, the proliferating superficial nephrons; in the cerebellum, the cells of the external granular layer; and in the testis, the Sertoli cells, which are at a critical period of development.

Cells dying in all three tissues showed the morphological features of apoptosis, a distinct form of cell death that has been identified in mammalian tissues, under physiological as well as pathological conditions. In contrast to the degenerative phenomenon of necrosis, there is evidence that apoptosis involves active cellular self destruction. Apoptosis has previously been reported following irradiation of rapidly proliferating adult cell populations.

It is suggested that activation of apoptosis might bring about selective elimination of cells with critical DNA damage in irradiated tissues, thus minimising propagation of genetic errors. This would obviously be particularly important in young, rapidly growing animals.

DNA TOPOISOMERASES TYPE I & II IN ATAXIA-TELANGIECTASIA

by

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Abstract

DNA topoisomerases are enzymes that alter the conformation of DNA by introducing transient breaks into DNA. These enzymes have been divided into two classes, type I and type II. They are distinguished by their reaction mechanisms.

Since the anomaly in DNA replication in cells from individuals with the human autosomal recessive disorder ataxia-telangiectasia (A-T) could be explained by change in chromatin structure or recognition of this structure we have assayed for possible defects in topoisomerases in these cells.

The results demonstrate that reduction in topoisomerase I and topoisomerase II occurs in A-T cells. Some variability was observed in different A-T cell lines. We are investigating these reduced activities by purifying topoisomerases.

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G2 PHASE DELAY IN ATAXIA-TELANGIECTASIA AND CONTROL CELL TYPES

by

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Abstract

Painter and Young (1) have suggested that the radiosensitivity in cells from ataxia-telangiectasia (A-T) patients is due to failure to undergo radiation-induced delay which in normal cells provides time for repair of damage. Recent reports from this and other laboratories, using flow cytometry, have demonstrated a marked accumulation in G2 phase after exposure of A-T cells to ionizing radiation (2-5). The duration of the block in G2 phase was considerably greater in A-T cells, and no effect or a reduced effect on progression through other phases of the cell cycle was observed (2,3,6). On the other hand Lucke-Huhle (7) failed to describe a greater or more prolonged G2 phase block in irradiated A-T fibroblasts compared to controls using the same technique.

In this study we have extended an investigation of the G2 block in A-T cells to include 8 A-T lymphoblastoid cell lines. Flow cytometry has been used in an attempt to distinguish between irradiated A-T homozygotes, A-T heterozygotes and controls.

All ataxia-telangiectasia homozygotes showed a significantly greater number of cells in G2 phase, 24h post-irradiation, than observed in controls. A more prolonged delay in G2 phase after irradiation was seen in different ataxia-telangiectasia cell types that included lymphoblastoid cells, fibroblasts and SV40-transformed fibroblasts. This technique appears to provide a reliable means of detecting radiosensitive cells, in this case ataxia-telangiectasia cells. However tha assay was incapable of distinguishing ataxia-telangiectasia heterozygotes from controls.

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CELLULAR STRESS INDUCED BY UV-RADIATION AND HYPERTHERMIA- A COMMON MECHANISM

by
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Abstract

All organisms from the simplest bacteria to plants to mammalian cells respond in a remarkably similar manner to abrupt changes in their environment. This apparent defensive mechanism, originally termed the heat-shock response, is now more commonly referred as the stress response due to the wide variety of diverse agents that induce basically the same cellular changes as those observed after a heat stress. Inducers of the stress response include heavy metals, ethanol, viruses and oxidative stress. The basic feature of the stress response in all organisms is the rapid and transient synthesis of a small set of proteins, termed the heat-shock or stress proteins (1).

We have used yeast cells as an experimental organism to test the hypothesis that cellular stress induced by UV-radiation and hyperthermia may share a common mechanism. Exposure of yeast cells to a non-lethal heat shock (37°C/30 min), sufficient to induce stress proteins, leads to an increase resistance to UV-induced cell death. Furthermore, heat-stressed cells are capable of recovery, following a period of incubation prior to plating, reminiscent of liquid-holding following radiation damage (2).

Ubiquitin is a 76-amino acid polypeptide which may be the most highly evolutionary conserved eucaryotic protein (3) and whose synthesis is induced by heat-shock. It is involved in protein modification reactions associated with removal and repair of damaged or abnormal proteins (4). Radiation, either as UV- or ionizing (γ, X-rays), leads to DNA damage and associated protein damage. We speculate that stress induced by radiation and hyperthermia leads to protein damage and/or formation of abnormal proteins. These moieties in turn act as eucaryotic stress signals whereby cells respond to stress by removing or repairing damaged/abnormal proteins via the ATP-dependent proteolytic cleavage ubiquitin system. Inability or improper repair of damaged proteins in stressed cells may lead to irreversible cellular injury and eventual cell death.

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EVIDENCE FOR METHYLATION INSTRUCTED MISMATCH REPAIR IN THE CHROMOSOMAL DNA OF CHO CULTURED CELLS

by

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Abstract

Using subclones of CHO cells having about 75% of the CpG methylation of the parental cell line we provide evidence for methylation - instructed mismatch repair in these cells.

ISOLATION AND CHARACTERIZATION OF HLA-A2 MUTANT CELLS

by

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Abstract

We have developed a technique for the study of mutations in human somatic cells at the HLA-A2 locus. This presents the opportunity to study the basis of mutations at an autosomal locus which is not possible with existing X-linked HPRT mutation systems.

In this work normal, X-ray or mitomycin exposed human peripheral blood lymphocytes from heterozygous HLA-A2 individuals were treated with a monoclonal anti-HLA-A2 antibody (BB7.2) and complement. Surviving cells were cultured at limiting dilution. Fourteen to 20 days later clones identified as positive wells in microplates were scored and were further tested with BB7.2 and complement. These clones were expanded and were retested by immunofluorescence using BB7.2 and other HLA-A2 antibodies and FITC conjugated anti-mouse antibody. Cells not expressing the HLA-A2 antigen were phenotypically analysed and DNA analysis using HLA-A specific DNA probes was performed.

The spontaneous mutation frequency determined for lymphocytes selected on day 0 was $2.59 \times 10^{-5} \pm 3.34 \times 10^{-6}$ (10 experiments), and for lymphocytes selected after 3-13 days in culture was $2.13 \times 10^{-5} \pm 2.93 \times 10^{-6}$ (30 experiments). Mutation frequency following exposure to X-ray or mitomycin C prior to selection showed a dose response.

HLA typing showed that 188 of 192 clones studied had lost the HLA-A2 antigen; the 4 clones which were HLA-A2$^+$ were excluded from the study. Both HLA typing and DNA analysis were performed in a total of 119 clones. Gene deletion was observed in 70 of these clones and simultaneous loss of an HLA-B allele was observed in 35 of these 70. Gene deletion was not observed in 49 clones and both HLA-B alleles were detected phenotypically in 47 of these 49. One clone showed a novel band by Southern analysis.

Gene deletions at the HLA-A2 locus and associated phenotypic changes at presumptively linked HLA-B loci were correlated with the mutagenizing agent used. Mitomycin C appears to produce large deletions as 23 of 31 clones showed loss of an HLA-B phenotype. Fourteen of 18 clones showed loss of the HLA-A2 gene by DNA studies. In contrast, X-radiation appears to produce smaller deletions evidenced by 21 of 27 clones showing loss of the HLA-A2 gene whereas only 4 of 29 clones so produced showed loss of an HLA-B allele phenotypically. Spontaneous mutants were characterized by changes intermediate to the results above.

The combination of gene and phenotypic changes is a powerful approach to characterizing mutants at the HLA loci.

RADIATION SENSITIVITY OF HUMAN NATURAL KILLER CELLS RECOGNISING BONE MARROW TARGETS

by

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ABSTRACT

Natural killer (NK) cells are spontaneously cytotoxic towards a number of neoplastic, and some normal cells. Their presence in the peripheral blood of all higher vertebrate species studied to date has suggested a role in anti-tumour surveillance. More recently, however, they have been implicated in the control of haemopoietic differentiation and consequently in the success or otherwise of bone marrow transplants.

NK cells are known to be relatively radio-resistant, and their activity (against standard leukaemic targets) has been reported to be increased by low levels of X- or γ-irradiation. The present study has revealed no evidence of this stimulatory effect of irradiation and has been designed to determine instead, whether bone-marrow active sub-populations of NK cells might be recognised by differential susceptibility to various irradiation regimes. The results of a series of irradiation doses, using standard cytotoxicity or cold-target competition assays, and a panel of normal bone marrow target populations will be presented.

MICRONUCLEI: AN IMPORTANT END-POINT IN RADIATION BIOLOGY

by

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Abstract

Micronuclei provide a measure of chromosome damage because they originate from chromosome fragments or whole chromosomes which are not included in the daughter nuclei of a dividing cell. Conventionally micronuclei are scored in all cells regardless of their nuclear or cell division status with the result that the micronucleus frequency ratio obtained is imprecise as it will vary with the proportion of cells that have completed nuclear division.

The correct procedure is to score micronuclei only in those cells that have completed one nuclear division. Recently an important modification to the micronucleus assay was developed that allows these cells to be identified [1]. In the new technique, the cytokinesis-block method, cells that have completed one nuclear division are recognised by their binucleate appearance following inhibition of cytokinesis by cytochalasin-B and micronuclei are scored in these cells alone. The results obtained by this method are not confounded by varing cell kinetics.

The precision and statistical power of the new method has resulted in increased sensitivity such that in vitro exposure to an acute dose of 2cGy (1cGy = 1 rad) X-rays can be detected. The cytokinesis-block micronucleus assay is a simpler and more sensitive method than chromosome analysis and should therefore prove to be a better tool for population screening. Results from our current longitudinal studies on micronucleus frequencies in lymphocytes of (a) cancer patients undergoing fractionated partial-body irradiation and (b) BALB-C mice following single-dose whole-body irradiation will be discussed.

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OUTLINE OF THE PROPOSED RADIOECOLOGY STUDY AT THE MARALINGA WEAPONS TEST SITE

by

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Abstract

An outline of the extent of contamination at various test sites at Maralinga resulting from the different operations carried out there will be presented. Possible hazards to members of the public which could arise from this contamination and the proposed program to delineat the possible exposure to radioactivity which may come from the ingestion pathway, with particular reference to aboriginal food items, will be detailed.

IN VITRO RADIOSENSITIVITY OF HUMAN MALIGNANT MELANOMA CELLS TO LOW AND HIGH LET RADIATION

by

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Abstract

Three human malignant melanoma cell lines have been characterised by measurement of plating efficiency, cell cycle parameters and generation times, cell density at confluence, chromosome constitution, melanin content and uptake of boron following a 20 hour incubation with ^{10}B-boronphenylalanine at 10 μg mL^{-1}.

Shouldered survival curves were observed following graded doses of X- or γ-rays for two cell lines with a D_o ∿1.8 Gy. Exponential survival curves were obtained for thermal neutron and γ exposures in the Moata reactor beam. Small but significant differences in survival were found for two cell lines with and without pre-incubation with ^{10}B-boronphenylalanine, the D_o values being 1.15 and 1.0 Gy respectively.

FAST NEUTRON IRRADIATION FACILITIES AT ANSTO - SNIF

by

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Abstract

Neutrons are an important tool for radiobiology studies, however they have had limited use due to the lack of suitable sources. Neutrons produced by radioactive sources usually have a poorly characterised fixed energy spectrum, low fluxes and possess a high gamma background. These factors complicate the interpretation of results and can make dose calculations difficult. The large gamma fluxes accompanying reactor neutrons and the fixed energies from D-T neutron generators also present problems.

The Standard Neutron Irradiation Facility SNIF at Lucas Heights comprises several beamlines on the 3 MV Van de Graaff accelerator dedicated to producing neutron beams with well characterised neutron energies, fluxes and angular distributions. Monoenergetic and broad spectrum neutron beams with energies from tens of keV to many MeV can be produced using reactions such as ^{7}Li+p, ^{3}T+p, ^{2}D+d, ^{3}T+d and ^{9}Be+d. By choosing appropriate targets and bombarding particles, the neutron energies and fluxes produced may be tailored to some extent to meet user requirements. Most of the neutron producing reactions are relatively free of any associated gamma fluxes.

The addition of a deuterium gas target to SNIF has enabled the production of high fluxes of monoenergetic neutrons up to 6 MeV using the D+d reaction. Fluxes of up to approximately 3×10^{8} neutrons $cm^{-2}sec^{-1}$ corresponding to a dose rate of approximately 50 Gy per hour have been produced. The maximum neutron fluxes available in any of the reactions is usually set by target heating effects created by the bombarding charged particles from the 3 MV Van de Graff accelerator.

For many biological applications, it is necessary to obtain from the neutron energy spectrum and fluence measurements the absorbed sample dose. This is possible by using a variety of computer codes on the SNIF database which take into account the finite geometry, reaction kinematics, sample composition and size.

SAMARIUM - 153 CHELATE LOCALIZATION IN MALIGNANT MELANOMA

by

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Abstract

Samarium - 153, a radiolanthanide of half life 46.27h, has a gamma emission of 0.103 MeV which is suited to imaging. ^{153}Sm is a moderate energy beta emitter and tumour localization of various ^{153}Sm chelates has been evaluated in B16 murine melanoma to assess their endoradiotherapeutic potential.

^{153}Sm was prepared from enriched ^{152}Sm in the Australian Nuclear Science and Technology Organisation research reactor. ^{153}Sm chelates were prepared from ^{153}Sm chloride and their chromatographic behaviour characterized. Tumour and organ uptake of ^{153}Sm chloride, ^{153}Sm chloride, ^{153}Sm citrate and the ^{153}Sm chelates DTPA, HEDTA, HIDA, BZ, PBH, PIH and NTA were measured at 1, 6, 24 and 48h after intravenous administration to C57 black mice bearing either melanotic or amelanotic B16 melanoma of mean size 0.75 cm^3.

^{153}Sm chloride was immobile on chromatography and the rapid hepatic accumulation of both ^{153}Sm chloride and ^{153}Sm citrate was attributed to in vivo formation of a colloid. In contrast ^{153}Sm-DTPA, moving at the solvent front on chromatography, showed no reticuloendoethelial accumulation in vivo and was rapidly excreted by the kidneys without tumour uptake.

The other ^{153}Sm chelates were of intermediate stability and all localized in both melanotic and amelanotic tumours, although to a significantly lesser degree than ^{67}Ga citrate. The relatively high ^{153}Sm-HIDA activity in liver and ^{153}Sm-NTA activity in bone impaired tumour definition but on imaging of all the ^{153}Sm chelates only ^{153}Sm-DTPA failed to demonstrate the B16 melanoma and the best tumour delineation was obtained using ^{153}Sm-HEDTA.

AUSTRALIAN RADIOISOTOPES PRESENT AND FUTURE: A PRESENTATION OF THE AUSTRALIAN MANUFACTURE OF RADIO-PHARMACEUTICALS FOR NUCLEAR MEDICINE

by

K.W. Horlock

Australian Nuclear Science and Technology Organisation,
Commercial Products Unit,
Lucas Heights Research Laboratories
Private Mail Bag 1, Menai, 2234

37R

Session VII Tuesday 3.50 - 4.20 p.m.

GENERAL INFORMATION

GENERAL INFORMATION

VENUE

The Conference will be held in the AINSE Theatre (Institute Bldg.) - see map page 47, on Monday 24th August, 1987 and Tuesday, 25th August, 1987.

PAPERS

Timing

Green light shows for presentation of paper,
Warning lights show when 5 & 2 minutes are remaining,
Red light shows when presentation time has expired,
Discussion time of 5 minutes is then allowed by the Chairman.

Slides

Authors using 35 mm slides in conjunction with their talk are requested to place their slides in the projector magazine during the break preceding the session in which the paper is scheduled.

Poster Session

The Poster Session will be held in Stevens Hall and Dining Room at the scheduled time. Posters should be set up before the Poster Session commences and removed after the final session that day.
Posters should be prepared before arrival at the conference in accordance with guidelines previously provided.
Authors are expected to be in attendance by their posters throughout the poster session.
Materials for setting up posters are available from any member of the AINSE staff.

ACCOMMODATION

For out of Sydney participants whose nominations have been previously accepted, accommodation has been arranged in accordance with advised requirements, at Stevens Hall, Lucas Heights (adjacent to the Institute Building, outside the Main Gate) - see map page 47, or at the Sapphire Motel, 408 Princes Highway, Sylvania Heights, see map back cover. The Institute will make payment directly to the managements for room only charges at Stevens Hall or the Sapphire Motel. Participants should make personal arrangements to pay cash for breakfast charges at Lucas Heights and the Sapphire Motel. Participants are requested to vacate Stevens Hall rooms by 9.00 a.m. Tuesday, and to leave their luggage in the room marked 'luggage' adjacent to the theatre foyer. Room keys to be left in the Stevens Hall Reception Office.

MEALS

Breakfast

For Stevens Hall residents, breakfast is served in the ANSTO Canteen from 7.30 a.m.
For Sapphire Motel residents, breakfast time to be advised.
Participants should make arrangements to pay cash for breakfast charges at Lucas Heights or the Sapphire Motel.

MEALS

Conference Lunch - Monday, 24th August, 1987

Lunch for all participants will be held in the Stevens Hall dining room during the scheduled lunch period (ref. programme). The cost has been included with the conference dinner payment.

Lunch - Tuesday, 25th August, 1987

Lunches may be purchased from the ANSTO Canteen.

Conference Dinner - Monday, 24th August, 1987

Pre-Dinner Drinks - Stevens Hall Lounge, with Poster Session
6.15 p.m. Buffet Dinner - Bamboo Room, ANSTO Canteen
7.45 p.m. Conference Address - Professor Sir Ernest Titterton, A.N.U.

Note No other meal service will be available at Lucas Heights on this evening.

Evening meals are provided on any other evening in Stevens Hall Dining Room, from 5.30 to 6.00 p.m. for guests of Stevens Hall, and the Sapphire Motel. Please advise the Conference Secretary if you require an evening meal on Sunday, 23rd August, 1987.

TRANSPORT

Transport from Sydney Airport - Monday, 24th August, 1987

An ANSTO bus will leave the AA Terminal (Sydney Airport) for Lucas Heights at 9.25 a.m. Kindly give your name to the driver when entering the bus. If any difficulty is experienced in locating the bus, the AA Commonwealth Car Desk will advise details of its precise location.

Request to Participants Arriving on ANSTO Bus from Airport

As time will be limited, participants are asked to go directly to the AINSE Theatre and not to their rooms at Stevens Hall. Luggage may be left in a room adjoining the theatre foyer, marked 'Luggage'. Keys may be picked up from Stevens Hall Reception at any time during the day.

Transport from Sydney Airport (exluding Special arrangements for Monday 24th August, 1987)

Taxis are available from the airport to:-
Lucas Heights,
Sydenham Railway Station - then train to Sutherland Station,
Sutherland Railway Station - then bus to Lucas Heights, or taxi (ref. bus timetable below).

Note:-
Bookings must be made for all transport listed below - through AINSE, as transport is only provided if demand exists.

Monday - Friday

Depart Lucas Heights for Sutherland	Depart Sutherland for Lucas Heights
7.35 a.m.	* 7.55 a.m.
8.30 a.m.	* 8.15 a.m.
9.35 a.m.	9.05 a.m.
10.35 a.m.	10.05 a.m.
11.30 a.m.	11.10 a.m.
12.30 p.m.	12.00 p.m.
1.00 p.m.	1.10 p.m.
2.15 p.m.	1.30 p.m.
3.35 p.m.	2.50 p.m.
* 5.00 p.m.	4.00 p.m.
8.20 p.m.	8.40 p.m.
9.20 p.m.	9.40 p.m.
10.25 p.m.	11.00 p.m.
11.35 p.m.	12.05 a.m

Buses from Lucas Heights leave from outside ANSTO Main Gate.

Buses from Sutherland leave from outside Post Office, Flora Street.

* Upon request these buses will transport participants to the Sapphire Motel.

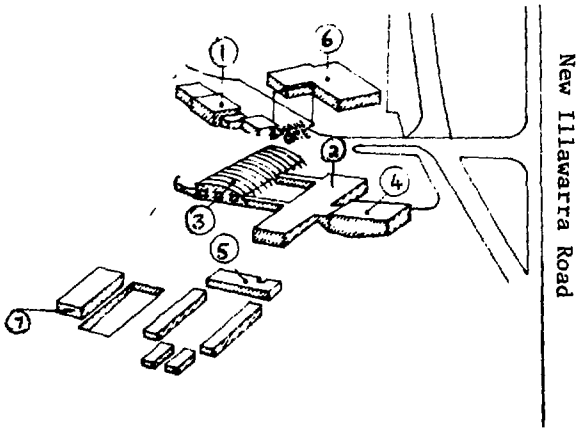
Saturday

Depart Lucas Heights for Sutherland	Depart Sutherland for Lucas Heights
6.30 a.m.	6.55 a.m.
7.35 a.m.	8.00 a.m.
8.35 a.m.	9.00 a.m.
9.30 a.m.	10.00 a.m.
	3.00 p.m.
3.35 p.m.	4.00 p.m.
10.30 p.m.	11.00 p.m.
11.35 p.m.	12.05 p.m.

Sunday

Depart Lucas Heights for Sutherland	Depart Sutherland for Lucas Heights
7.35 a.m.	8.05 a.m..
8.35 a.m.	9.00 a.m.
2.20 p.m.	2.50 p.m.
3.35 p.m.	4.00 p.m.
4.30 p.m.	5.00 p.m.
10.20 p.m.	10.50 p.m.
11.35 p.m.	12.05 a.m.

LUCAS HEIGHTS N.S.W.



1. Main Gate
2. AINSE Building
3. ANSTO Canteen
4. AINSE Theatre
5. Stevens Hall
6. Reception
7. Amenities Centre

Transport from Sapphire Motel to Lucas Heights

Transport will be arranged for participants. Please wait outside the motel reception office for pick-up at the following times:-

Monday, 24th August, 1987	8.45 a.m.
Tuesday, 25th August, 1987	8.20 a.m.

Transport from Lucas Heights to Sapphire Motel

Buses leaving Lucas Heights at the following times will transport participants to the Sapphire Motel - 8.20 p.m., 9.20 p.m. and 10.25 p.m. (see timetable). It is necessary, however, to book for these services - through AINSE, as transport is only provided if demand exists.

Transport from Lucas Heights to Sydney Airport - Tuesday, 25th August, 1987

Transport will be arranged to take participants to Sydney Airport after the conference concludes. Please place your name on the list provided, on the noticeboard outside the theatre, if you require this service.

Participants leaving Stevens Hall - Tuesday, 25th August, 1987

Participants are requested to vacate their rooms by 9.00 a.m. Luggage may be left in the room marked 'Luggage' adjacent to the AINSE theatre foyer. Room keys may be left in the Stevens Hall Reception Office.

TELEPHONE MESSAGES

Telephone messages will be taken for conference participants on:-

543-3411 (AINSE)	543-3436 (AINSE)	543-3111 (Switchboard)

All enquiries concerning the conference arrangements should be directed to:-

Mrs. Joan Watson,
Conference Secretary,
A.I.N.S.E.,
Private Mail Bag 1,
MENAI NSW 2234

Phone: 543-3411 or 543-3436

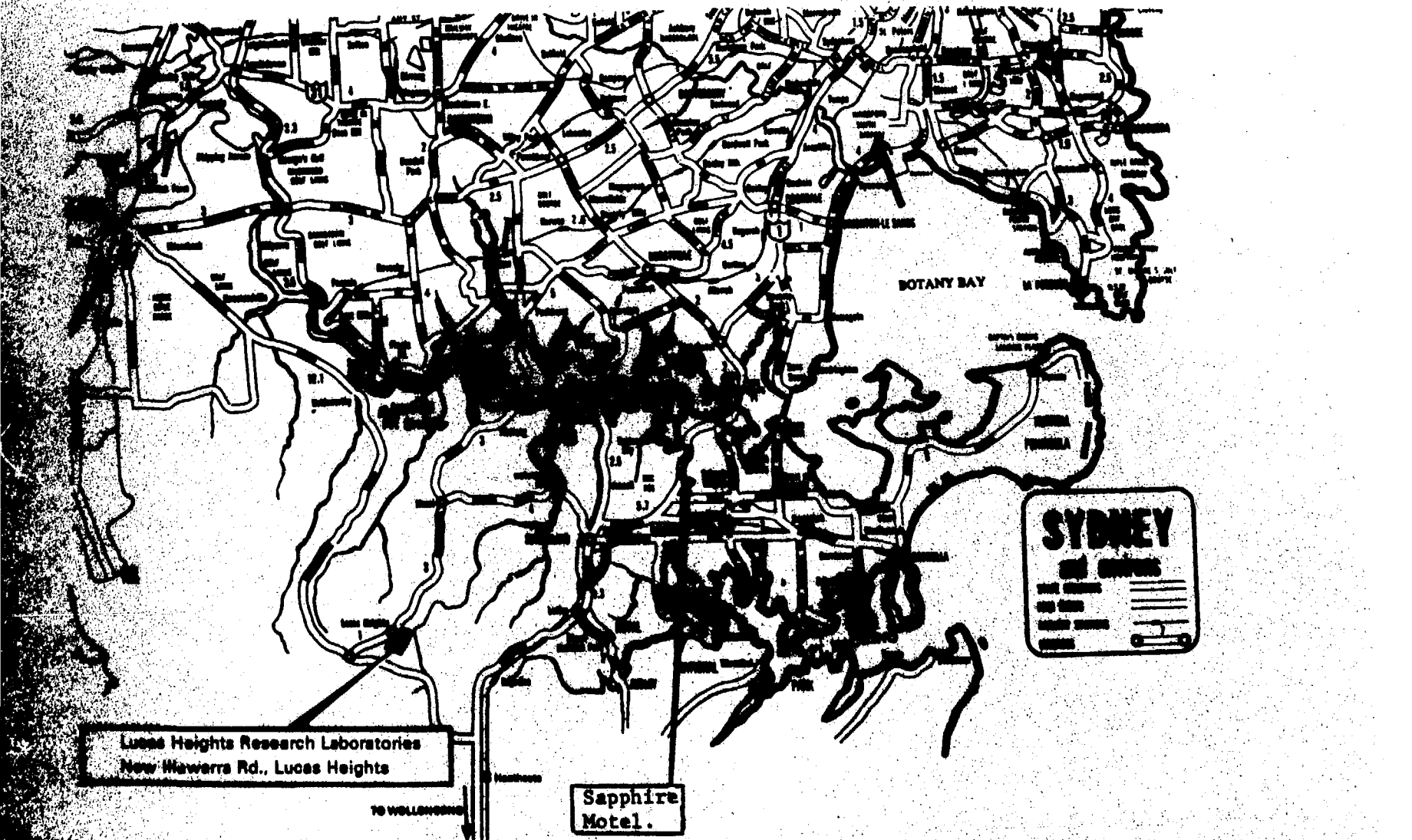
LIST OF PARTICIPANTS

PARTICIPANTS

OVERSEAS VISITORS		PAPER NO
Dr. R.D. Combes	Portsmouth Polytechnic, U.K. (Visiting La Trobe Univ.)	
Dr. P.B. Roberts	Inst. of Nucl. Sci., N.Z.	10R
UNIVERSITY OF QUEENSLAND		
(Department of Biochemistry)		
Dr. M.F. Lavin		7R,12,26,27,35
Dr. S.P. Singh		12,26
Mr. P. Bates		27
(Department of Pathology)		
Mrs. G.C. Gobé		25
UNIVERSITY OF NEW ENGLAND		
Department of Microbiology)		
A/Professor K. Watson		28
UNIVERSITY OF NEW SOUTH WALES		
(Centre for Safety Science)		
Dr. E.M. Nicholls		22
Dr. R. Rosen		
Mr. B. Markovic		
ANSTO		
(Environmental Science Division)		
Mr. D.R. Davy		
Mr. J.K. Brown		21,23R,24
Mr. M. Giles		33
Dr. R. Jeffree		13
Dr. H. Panter		14
Ms. B. Izard		
Mr. M.H. Mountford		21,34
Mrs. J. Chapman		14
(Health and Safety Division)		
Mr. J.C.E. Button		
(Applied Physics Division)		
Dr. J.W. Boldeman		12,35
Dr. B.J. Allen		5,15,21,34
Dr. N. Dytlewski		12,35
(Isotope Division)		
Mr. R.E. Boyd		36
Ms. P. Wills		9R
(Commercial Products Unit)		
Mr. K.W. Horlock		37R

	PAPER NO
CSIRO	
(Division of Molecular Biology)	
Dr. D. Podger	4
(Division of Plant Industry)	
Dr. R.D. Brock	
(Division of Food Research)	
Dr. M.A. Brown	
AINSE	
Dr. D.D. Cohen	12,35
AUSTRALIAN NATIONAL UNIVERSITY	
(Department of Nuclear Physics)	
Professor Sir Ernest Titterton	
UNIVERSITY OF MELBOURNE	
(Physical Safety & Radiation Protection Unit)	
F.P.J. Robotham	11R
(Department of Genetics)	
Dr. B.T.O. Lee	
Ms. J. Williams	
MONASH UNIVERSITY	
(Department of Anatomy)	
Dr. D.H. Barkla	21
LA TROBE UNIVERSITY	
(Department of Genetics & Human Variation	
Professor P.A. Parsons	
Dr. G.R. Flannery	31
(Department of Microbiology)	
Dr. D.G. MacPhee	1R,2,3,16, 17,18,19,20
Mr. T. Shaw	17,19,20
Ms. K. Silburn	3
Miss V. Bullwinkle	
Ms. J. D'Costa	
Ms. G. Grammaticopoulos	
Miss L. Hafner	2
Ms. D. Liaskou	
Ms. J. Williams	16
UNIVERSITY OF TASMANIA	
(Department of Agricultural Science)	
Dr. T. A. McMeekin	

UNIVERSITY OF ADELAIDE	PAPER NO
(Department of Zoology)	
Mr. M.J. Tyler	
(Department of Physical and Inorganic Chemistry)	
Dr. G.S. Laurence	6R
FLINDERS UNIVERSITY OF S. AUSTRALIA	
(Flinders Medical Centre, Department of Haematology)	
A/Professor A.A. Morley	30
Dr. M. Fenech	32R
Mr. M. Janatipour	30
MURDOCH UNIVERSITY	
(School of Mathematics and Physical Sciences)	
Dr. J. Webb	36
QUEENSLAND INSTITUTE OF TECHNOLOGY	
(Department of Physics)	
Dr. B.W. Thomas	
Dr. B.J. Thomas	
PETER MACCALLUM CANCER INSTITUTE	
(Molecular Science Group)	
Dr. R.F. Martin	5
Dr. W. Diver	29
Dr. L.S. Denison	
Dr. I. Radford	24R
Mr. G. d'Cunha	5
Mr. M. Pardee	5
Ms. D. Cheung	
QUEENSLAND DEPARTMENT OF PRIMARY INDUSTRIES	
(Food Research and Technology Branch)	
Dr. H.S. Juffs	
COMMONWEALTH DEPARTMENT OF ARTS	
(Nuclear Section)	
Dr. J.A. Ross	
RADIATION HEALTH SERVICES	
(Department of Health, N.S.W.)	
Mr. A. Fleischmann	
Mr. P. Colgan	
AUSTRALIAN CONSUMERS' ASSOCIATION	
Ms. S. Heilpern	8R
Dr. G.M. Watson	


BOTANY BAY
SYDNEY
Lucas Heights Research Laboratories
New Illawarra Rd., Lucas Heights
Sapphire
Motel.