Laureate and Thomas got the Nobel prize for his work in bone-marrow transplants.

The key question is not who publishes where and how often but whether a claimed authorship is justified. A scientific work is far better judged by not where it is published but by how often it is cited and how the work is followed up by others. Not all papers published in *Nature* become classics.

Today there is increasing awareness that scientists need to keep the public, whose money they use, informed and educated of what they do. Science is no longer an insulated pursuit of knowledge. Society depends on science to develop technology and to increase its standards of living. Science forms inputs to government policy formulations which, in turn, are influenced by public opinion. Having accepted a democratic set up and huge public funding for scientific research, scientists have an obligation to keep the public abreast of scientific developments. To bring the intricacies of science to the non-specialist, even the common man, we need more people like the late Richard Feynman and not hecklers who seem to lack an understanding of the spirit of science in the 1990s, to write accurately for popular magazines and newspapers. Indeed many media publications have science sections simply because there is a demand for it from the lay readers. Fortunately, CSIR's current policy is sympathetic to this idea as are all enlightened governments around the world.

The scientific community generally knows how to zero in on important publications. To cite a recent example, a very interesting paper in CFD (computational fluid dynamics) which appeared in the *Journal of Computational Physics*, was favourably reported upon in *Nature*. However, it is doubtful that CFD people would ever seriously consider publishing their work in *Nature* notwithstanding *Nature*'s preeminent status among scientific journals. It, however, speaks volumes about the editorial policy of *Nature* that in the true spirit of science it monitors the rest of the scientific publications world to inform its readers of important developments, including political, economic, and military.

The hallmark of a true scientist is to judge a paper on the basis of its scientific contribution rather than its packaging. Unfortunately we have encouraged the 'me and no one else' type of scientists who find it expedient to judge the work of their subordinate colleagues by its packaging simply because they lack the confidence to judge scientific contributions on scientific merits. These very people, instead of resolving scientific differences according to the centuries old traditions of the scientific community, resort to character assassination in a manner more fitting to roadside thugs. The Indian scientific community needs to set up traditions whereby such undesirable elements amongst us are weeded out.

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NEWS

Adaptive response in radiation biology

A recent meeting* brought together about 250 scientists including both the seasoned radiation biologists, cancer epidemiologists, cytogeneticists and radiation physicists as well as young investigators in the relevant fields from all over the world to discuss the topic of stimulatory effects of radiation and adaptive response at low dose and low dose rates and the immune reaction of the living organisms against radiation damage.

Leonard Sagan (Electrical Power Research Institute, Palo Alto, USA) giving some details on the origin of radiation paradigm, stressed the necessity of epidemiological studies and research on the possible molecular mechanisms of radiation effects. An explanation on the enormous strides made in the estimation of the risk of cancer following ionizing radiation in the past four decades was given by William J. Schull (University of Texas Health Science Center, Houston, USA, and Permanent Director, Radiation Effects Research Foundation, Hiroshima, Japan). He discussed the completeness and accuracy of such studies and the uncertainty in dose response relationship due to confounding factors. He concluded that no risk model has strong biological basis and if ionizing radiation is a promoter in inducing tumour there should be a threshold for this.

B. L. Cohen (University of Pittsburgh, USA) spoke on the test of the linear-no-threshold theory of radiation carcinogenesis with special reference to radon in houses. Discussing the high dose data available from radon in mines in the US, Canada, Czechoslovakia and Sweden, he showed a decreased lung cancer in the population with an increase in radon dose. J. R. Maisin (Universite Catholique de Louvain, Bruxelles, Belgium) described the most promising treatments with single or in combinations of nontoxic doses of radioprotectors or biological response modifiers before exposure to ionizing radiation. He suggested that there is a need for new radioprotectors which are less toxic when given alone or in association with low levels of aminothiols.

*International Conference on Low Dose Irradiation and Biological Defence Mechanisms, Kyoto, Japan, July 12–16, 1992. The proceedings of the conference will be published by the Elsevier Science Publishers, Amsterdam.

Considering the current controversial topic of transgeneration carcinogenesis risk by radiation, the presentation by T. Nomura (Osaka University, Osaka, Japan) on *in utero* and transgeneration effects and biological defence mechanisms in mice is of immediate relevance. He explained the defence mechanism to toxic agents in the early developing embryos. He showed that apoptotic cell death at pronucleus stage during organogenesis may be the earliest defence mechanisms, thereby eliminating the pathogenic cells. Based on the classical experiments with mice, he stated that parental exposure to radiation could induce various kinds of tumours in the next generation.

H. Planel (Medical School, Toulouse, France) emphasized his presentation on radiation hormesis through his studies on animal models. His investigations showed that a chronic irradiation at very low dose rate, less than 1 cGy per year resulted in an hormetic effect. He said that activities of G6P-DH, glutathione reductase and superoxide dismutase are stimulated by the very low doses.

The possible molecular mechanisms of physiological system of the animal after exposure to ionizing radiation were also discussed (S. Liu, Institute of Radiation Medicine, Normal Bethune University of Medical Sciences, Jilin, China). The existence of a cellular defence system in various species, including the mechanisms such as antioxidation, DNA repair, ADP-ribose polymerization, stress protein induction, etc. was also discussed. According to him, the secretion of interleukins such as interleukin 2 (IL-2) and gamma interferon (INF-gamma) is increased and the proliferation of T-lymphocytes is potentiated after low dose irradiation. T. Makinodan (Geriatric Research, Education and Clinical Center, Los Angeles and Department of Medicine, University of California at Los Angeles, USA), in his presentation on the hormetic effects of low dose radiation, explained the molecular mechanisms behind this phenomenon. He suggested that enhancement of the response and down-regulation of tumour incidence after low dose radiation suggest that low dose radiation can act as a hormetic agent by modulating the antigen-stimulated clonal growth and/or the differentiation process of immune cells. A possibility that chronic low dose radiation can induce an homeostatic up-regulation in the repair of constitutive and radiation-induced DNA-strand breaks in splenocytes was first investigated by his group.

Sheldon Wolff (University of California, San Francisco, USA), speaking on low dose exposures and the induction of adaptation, hypothesized that when human lymphocytes are exposed to very low doses of X-rays (1 cGy) an adaptive response occurs that makes the cells less susceptible to the induction of chromosome breakage by subsequent exposures to high doses of X-rays (150 cGy). This adaptive response, which takes 4–6 h to develop fully and lasts for three cell cycles, has been attributed to the induction of hitherto unknown DNA repair mechanisms, which lead to the repair of more of the primary breaks in DNA. His experiments with two-dimensional gel electrophoresis have shown that 1 cGy of X-rays induces proteins, which are considered to be excellent candidates for being the enzymes responsible for repair. He has also stated that there is a *window* of dose below which and above which the proteins are not induced. G. E. Adams (MRC Radiobiology Unit, Didcot, UK) described different types of insults which can induce cellular stress response like heat, hypoxia, UV-light (360 nm), glucose deprivation, ionizing radiation and magnetic fields. In his talk he reviewed various aspects of the relevance of stress responses to cancer through data both from the published literature and from his own laboratory. He pointed out that there are several reports of stress-induced up-regulation of various oncogenes and genes whose expression is an early event during activation of cellular proliferation.

Hormesis is the phenomenon of induction of beneficial effects by low doses of otherwise harmful physical or chemical agents (J. Smith-Sonneborn, University of Wyoming, USA). She proposed the hypothesis that the common pathway for hormesis is a heat shock-like response or stress response. She reported the different methods to operate the hormetic response through stress response as: (1) identification of agents known to induce both the stress response and hormetic phenomena; (2) a description of the unique and common pathways in the stress response to three stressors, heat DNA damaging agents and teratogens; (3) the stress response as a model for teratogen-induced damage and (4) a theory for an explanation of the paradoxical beneficial response to low doses of an otherwise harmful agent via a stress-response pathway. M. M. Elkind (Colorado State University, USA) compared the cell sensitivity to neoplastic transformation for high- and low-LET radiation during different cell cycle timings.

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