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In This Issue...

Editorial Board

Time Trends in Cancer Incidence Rates 1982-2005

The National Cancer Registry Programme (NCRP) was established under the Indian Council of Medical Research in 1981 with an aim of looking at the incidence and distribution of cancer in the country. Three hospital based (HCRs) and 3 population based cancer registries (PBCRs) started data collection from January 1, 1982. Six more PBCRs and HCRs started functioning from 1988. Seventeen other PBCRs have started functioning in recent years. Since the commencement of the NCRP annual, biannual and consolidated reports have been regularly published. A brief picture on time trends in incidence rates was depicted in the consolidated report for the years 1990-96[1].

The first comprehensive report on Time Trends in Cancer Incidence Rates: 1982-2005 under the NCRP of the ICMR has been recently published (NCRP, 2009). The salient findings of this report has been summarized in the present write-up.

The report covers information for the twenty four year period (1982-2005) for the population based cancer registries at Bangalore, Chennai and Mumbai and for an eighteen year period (1988-2005) for the registries at Barshi, Bhopal and Delhi. Except, Barshi the registries essentially cover urban populations that display rapid life style changes.

A common question of interest is “Is Cancer on the Rise?”. Cancer is known to be a disease that increases in incidence with increasing age. The control of communicable diseases, has increased life expectancy and therefore exposed more of the population towards the development of cancer. The increase in population due to growth also contributes to the increase in the number of cancer cases. Improved literacy, greater consciousness about health in general and awareness about cancer in particular makes more and more people seek medical advice at an earlier stage. Availability of sophisticated and improved diagnostic techniques aid in detection of tumours, that could have been missed at earlier times. The question is whether cancer is on the increase after accounting for these factors and whether that rise is statistically significant.

One measure of determining such an increase would be to examine the age adjusted incidence rates (AAR) over time. This may or may not take into account

all of the factors mentioned above. Nonetheless, it would give some indication of the trends in the disease. Cancer being a chronic disease (and unlike infectious diseases) with generally a long latent period and a rather prolonged clinical phase, year to year variations are minimal. Therefore, in assessing time trends in AAR, the normal practice in registries across the world is to look at five yearly rates over decades. This would give a more definitive indication of the course of the disease. Nonetheless, the data presented here give a fair account of the direction in which the incidence rates of the leading sites of cancer are proceeding across the years. Based on this, the report also provides an estimate of the burden of specific sites of cancer for the next decade. Such estimates will greatly facilitate deciding on priorities and planning site specific cancer control activities.

Data Collection at PBCRs

Population based cancer registries collect information on cancer occurrence from all centres that are likely to see cancer patients from a different geographical area. Information on all cancer cases pertaining to calendar years is collected from the hospital / health setup case records and / or from the patients on a proforma specially designed for the NCRP of the ICMR. After removal of duplicates, the data for different calendar years is sent to coordinating unit for possible cleaning and analysis.

Method of Calculating Time Trends

The numerator data of all registries have undergone a series of range and consistency checks each year and again before preparing the report. Clarifications were sought wherever required from the respective PBCRs and the data finalised thereafter. The difference distribution method[2] for estimating the calendar year wise denominator population by five year age group has been used. This is based on the census data of 1981, 1991 and 2001. The age specific data are standardized to world population (as provided by the International Agency for Cancer Research, World Health Organization). Such age standardized incidence rates help in direct comparison of data (to adjust variations in age of populations) between registries over time for the same area. In determining the significance of trends, the actual value of the AAR for each year has been used[4]. The significance of time trend in each PBCR was assessed based on the methods and formula provided by Boyle and Parkin[3]. In addition, the Joinpoint Regression Programme of the National Cancer Institute (NCI) of USA has been used. Joinpoint Regression Programme, Version 3.0, is a statistical software for the analysis of trends using Joinpoint models, that is, where several different regression lines are connected together at the "Joinpoints". Cancer trends reported in NCI publications are calculated using the Joinpoint Regression Programme to analyze rates calculated by the Surveillance Epidemiology and End Result (SEER). The combination of time trend in pooled crude rates of selected sites for past five years and population estimate by time was used to arrive at the projection

The summary of the results are given in table I.

There has been a steady and consistent increase in the AARs of certain cancers across all major urban

TABLE I: Values of annual percentage change for those anatomical sites of cancer that have shown statistically significant change

Site	ICD-10	Bangalore	Chennai	Delhi	Mumbai
MALES					
Colon	C 18	2.2	2.2	1.7	-
Rectum	C 19-20	1.6	2.5	-	-
Liver	C 22	2.0	1.6	1.7	1.9
Prostate	C 61	2.4	4.7	3.1	0.8
Brain	C70-72	3.2	3.0	-	2.7
NHL	C 82-85, C96	1.5	2.5	1.0	1.9
Lymphoid leukaemia	C 91	2.7	2.6	-	-
Myeloid leukaemia	C 92-94	2.2	2.5	-	0.8
FEMALES					
Gall Bladder	C23-24	5.9	5.9	-	2.5
Lung	C 33-34	2.7	4.6	2.0	-
Breast *	C 50	2.7	2.5	1.0	1.2
Cervix **	C 53	-2.6	-3.2	-3.4	-1.7
Corpus Uteri	C 54	5.8	2.2	3.8	1.7
Ovary	C 56	1.7	-	-	0.8
Brain	C 70-72	3.4	4.6	-	2.9
Thyroid	C 73	-	2.6	2.2	-
NHL	C 82-85, C96	3.5	2.8	1.2	1.8
Myeloid leukaemia	C 92-94	1.9	3.4	-	-

*Registry at Bhopal has also shown a significant increase (APC 1.4)
**Registries at Barshi and Bhopal have shown a significant decrease (APC Barshi 2.2 Bhopal 4.1)

registries. Among males, cancers of the prostate, colon, rectum and liver, have shown statistically significant increase in incidence. Cancer of the prostate is the leading site of cancer among males in most of the western countries as is cancer of the colon. Among females, cancers of the breast, corpus uteri and lung have shown a rise. Three other sites of cancer that have shown an increase in incidence rates in women are ovary, thyroid and gallbladder. Both males and females have recorded rising incidence rates for cancers of the brain as well as in tumours of the lymphoid and haemopoetic system, especially non-Hodgkin's Lymphoma. The Joinpoint regression trend graphs on some selected sites of cancer are depicted in figure 1-5.

A decline in the incidence of cancer cervix is seen across all registries including the rural registry at Barshi. This decline is observed in the absence of any organized screening or early detection programmes in the registry areas.

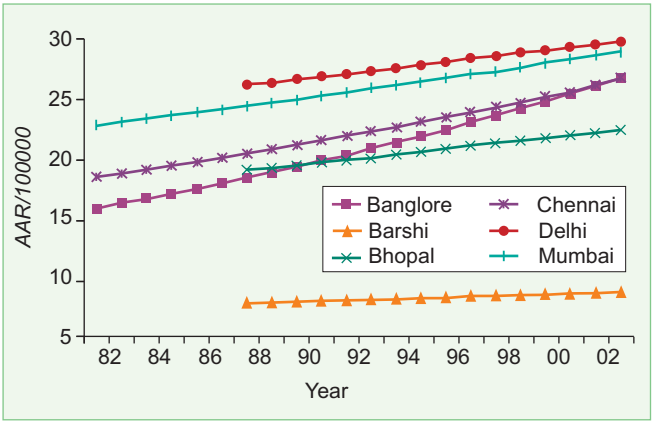

Fig 1. Joinpoint regression Model trend lines – Breast (females)

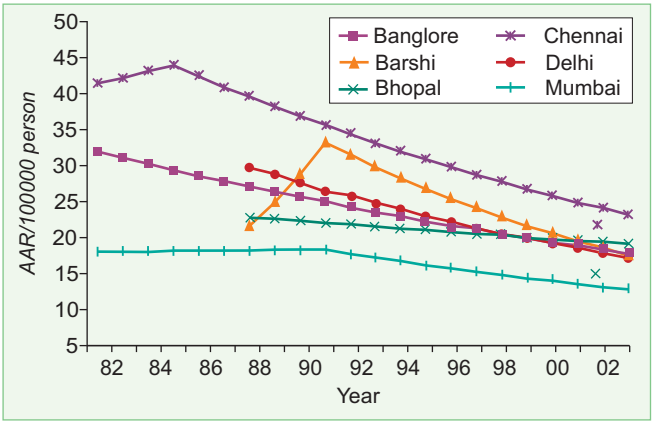

Fig 2. Joinpoint regression Model trend lines - Cervix

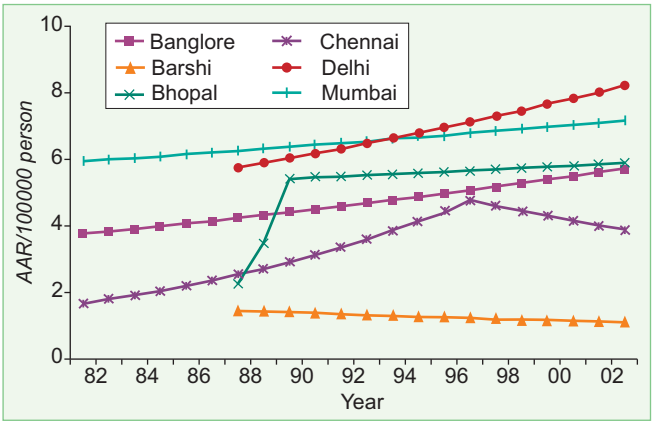

Fig 3. Joinpoint regression Model trend lines – Prostate (Males)

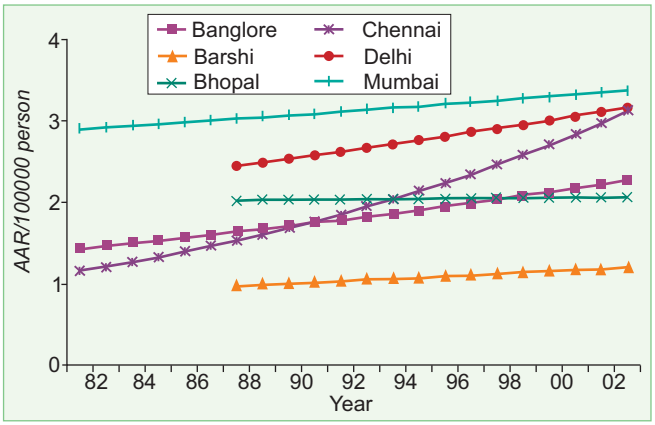

Fig 4. Joinpoint regression Model trend lines – Lung (Females)

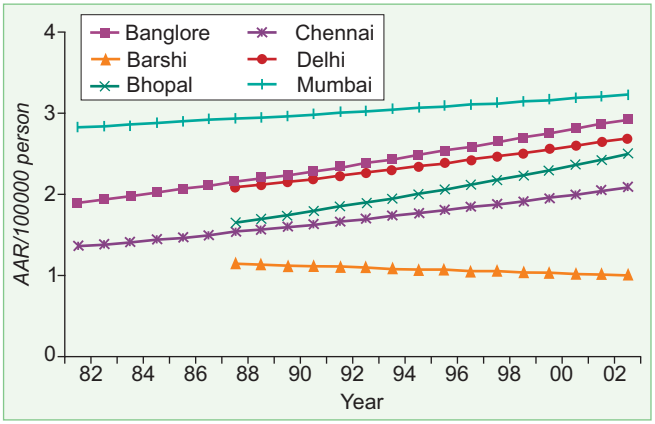

Fig 5. Joinpoint regression Model trend lines – Colon (Males)

Estimated Burden of Cancer

Projection of cancer burden means a systematic way of prediction of number of cancer cases for all

TABLE II: Cancer burden in India: Estimated new cancers
All anatomical sites (ICD-10: C00-C96)

Year	Males	Females	Total
2008	447399	498773	946172
2009	454842	507990	962832
2010	462408	517378	979787
2015	497081	563808	1060889
2020	534354	614404	1148758

anatomical sites or for a specific site and for a specified period of time. This could be based on time trend in incidence rates based on the projected population. The estimated new cancers are given in Table II.

Cancer Research and Control Opportunities

The cancer registry is central to any rational programme on cancer control. With information on trends over time, there appears to be a need for reassessing priorities for cancer control[5]. The overall thrust of the cancer control programme towards anti tobacco education / legislation and control of cancer cervix is largely unchanged. However, certain specific pointers emerge from this report.

The rate of increase in the incidence of cancer of the lung among women is glaring calling for systematic evidence based and focused anti-tobacco campaigns targeting the urban woman. Two other cancers in women that are increasing and for which, promotion of awareness for early detection is required are cancers of the breast and corpus uteri. Among males, nation-wide screening programmes for cancer of the prostate, colon and rectum are essential.

While control of tobacco use has to be constantly emphasised, the importance of obesity control and genital hygiene could be propagated. The necessity for early detection should be increasingly highlighted along with public education and self examination methods. The role of registries would ultimately be made applicable with overall trend studies as well as trends of stage at disease presentation.

In the present background, cancer research could comprise epidemiological research, basic laboratory research or cancer control research or a combination of these. Risk factors, such as tobacco and its association with specific anatomical sites of cancer are well known. However, in the Indian context some of the tobacco habits (like types of tobacco chewed, beedi smoking) vary in different parts of the country and India specific tobacco related cancer research is required. Besides, this report has shown several specific sites of cancer that are rising in incidence rates. These are cancers of the prostate, colon, rectum and liver in men and thyroid and gallbladder in women. In both sexes, tumours of the brain and nervous system and the lymphoid and haemopoietic malignancies have shown a rise. This calls for systematic epidemiological research preferably taking into account laboratory parameters for each of these sites of cancer. One of the outstanding features noted relate to the contrasting incidence of certain cancers in relation to geographic locations. Cancers of the gall bladder and thyroid are prominent examples. These provide unique opportunities for multi-centric case control studies which have to be systematized now. Similarly, there are no definitive directly replicable models for early detection / screening of cancers of the thyroid, gall bladder, rectum, liver, ovary and corpus uteri. The first four sites are country specific and research into various modules for cancer control need to be done.

Conclusions

There are international and standard guidelines for clinical staging and protocols for treatment of most cancers, including the ones that are showing an increase in this report. Their applicability in our settings needs detailed studies and investigations. This seems particularly important for cancers of the prostate, colon and rectum in men and for cancers of the breast, ovary, corpus uteri and thyroid in women.

References

1. Boyle, P. and Parkin, D.M. Statistical methods for registries – In: *Cancer Registration – Principles and Methods*, Eds. O.M. Jensen, D.M. Parkin, R. Maclennan, C.S. Muir and R.G. Skeet. *IARC Scientific Publications 95*, 1991.
2. *Consolidated Report of the Population Based Cancer Registries 1990-1996*: National Cancer Registry Programme (ICMR), Bangalore, 2001.
3. Kim H.J., Fay, M.P., Feuer, E.J. and Midthune, D.N. Permutation tests for joinpoint regression with applications to cancer rates. *Stat Med 19*:335, 2000 (correction: 20:655, 2001).
4. Muir, C.S., Demaret, E. and Boyle, P. The cancer registry in cancer control: An overiew. *IARC Scientific Publication 66*: 13-1985.
5. Takiar, R. and Shobana, B. Cancer Incidence rates and problem of denominators - A New approach in Indian Cancer Registries. *Asian Pacific J Cancer Prev 10*: 123, 2009.

This write-up is based on the report entitled "Time Trends in Cancer Incidence Rates 1982-2005, published by the National Cancer Registry Programme (ICMR), Bangalore.

ABSTRACTS

Some Research Projects Completed Recently

A study on nutritional status in patients with chronic pancreatitis

The study was carried out on fifty four patients (39 patients of tropical pancreatitis – TP; and 15 patients of alcoholic chronic pancreatitis – ACP) and equal number of healthy controls matched for age and sex to assess their nutritional status and explore its relationship with features of the disease that might contribute to under nutrition. The anthropometric measurements (AM) and study of faecal elastase were also carried out and correlated with clinical parameters.

The BMI (19.15 ± 3.29 vs 24.64 ± 1.54, $p < 0.001$), triceps skin fold thickness (9.57 ± 3.85 vs 12.48 ± 1.16mm, $p < 0.001$), mid arm circumference (24.49 ± 3.33 vs 29.90 ± 4.28 cm, $p < 0.001$) but not the waist-to-hip ratio (0.91 ± 0.1 vs 0.92 ± 0.07. $p < 0.05$) were significantly lower in patients than in controls. Undernutrition (BMI<18.5) was equally common in TP and ACP {17 (43.59%) vs 6 (40%), $p > 0.05$}. BMI and other AMs were similar in TP and ACP. The pre-morbid BMI was higher than the index BMI (20.69 ± 3.89 vs 19.15 ± 3.29 kg/m^2) in patients.

It was concluded that the undernutrition is common in CP, develops after the onset of illness, occurs equally commonly in TP and ACP and hence appears to be the effect rather than the cause of the former condition.

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Conformational study of *Taenia solium* metacestode proteins diagnostic for neurocysticercosis: Is there a role for unfolding to imrove antigencity?

T. solium cyst infections of the brain (neurocysticercosis) are predominantly solitary cyst infections in India. This poses difficulties for serological diagnostic assays that detect antibodies, as the levels are apparently too low for detection in many of these patients. Antibody detection immunoblot assays for solitary cysticercus granulomas are specific but only 60% sensitive. These tests use a fraction of lentil lectin specific *T. solium* cyst glycoproteins that contain 7 infection specific proteins of molecular weights 50, 38, 24, 21, 18, 14, 13 kDa, in different concentrations, as antigen. The study was undertaken to determine if altering the structure of *T. solium* proteins diagnostic for neurocysticercosis will improve their antigenicity and thereby their detection of cysticercal antibodies in neurocysticercosis patients.

It is known that conformational alterations of proteins result in increasing the numbers of exosed epitopes and this was attempted with the cyst antigens. Six of the *T. solium* infection specific glycoproteins of molecular weights 50, 38,24, 18,14, 13kDa were purified to homogenicity by lentil lectin chromatography and preparative SDS-PAGE. These proteins required extensive N-glycosylation and disulfide bonds to maintain their antigencity. Use of urea (5M) induced tertiary conformations of these proteins improved sero-detection on immune blots of cysticercus antibodies among patients with solitary cyst infections to 71% from 62% using antigens not treated with urea.

Providing a cocktail of these antigens in equal concentrations (50-150ng/ml) improved serological antibody detection for solitary cyst infections to 88% but decreased the detection of multi-cyst infections. This paradox requires further study. The sensitivity of antibody detection was 83% for patients with solitary cysticercus granulomas in dot blot assay using each pure antigen at equivalent concentrations of fucose (0.25 pmoles). This study showed that serological diagnostic assays for patients with solitary cysticercus granulomas improved by using unfolded cyst antigens, cocktails of antigens in defined protein concentrations and antigens equivalent in fucose.

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Publication

Prabhakarana, V., Rajshekhar, V., Murrell, K.D. and Oommen, A. Conformation sensitive immunoassays improve the serodiagnosis of solitary cysticercus granuloma in Indian patients. *Trans R Soc Trop Med Hyg 101*: 570, 2007

Study of the mechanism of action of novel resistance modifying agents overcoming multidrug resistance in cancer

In search of a suitable resistance modifying agent (RMA) capable of deactivating resistance causing protein (P-gp, MRP) and glutathione (GSH), a number of novel Schiffs bases and their metal chelates were synthesized and characterized. Copper (II) N-(2-hydroxyacetophenone) glycinate (CuNG) was observed to be the most potent in overcoming multi drug resistance (MDR). CuNG increased ROS generation and reduced MRP1 expression in drug resistant EAC/Dox cells while only temporarily depleted GSH. CuNG also modulated the activities of super oxide dismutase, catalase and glutathione peroxidase in different organs of mice bearing MDR-cells like EAC/Dox, sarcoma 180 and Lewis lung carcinoma and thereby reduced oxidative stress.

It was observed that the level of copper in the serum was significantly higher in MDR-cancer bearing animals and also in cancer-patients unresponsive to drugs. The level of serum copper might be considered a biomarker for treatment response. Intramuscular (i.m.) administration of CuNG (5 mg/kg body weight) completely resolved drug resistance in doxorubicin-resistant Ehrlich ascites carcinoma (EAC/Dox) bearing mice and doxorubicin-resistant sarcoma 180 bearing mice. CuNG-treatment resolved drug-resistant cancers through induction of apoptogenic cytokines, such as IFN-γ and/or tumour necrosis factor-α from splenic mononuclear cells or patient peripheral blood mononuclear cells (PBMC) and reduced the number of T-regulatory marker-bearing cells while increased infiltration of IFN-γ-producing T-cells in the ascitic tumour site. The potential usefulness of CuNG was proved in immunotherapy of drug resistant cancers irrespective of multidrug resistance phenotype.

CuNG treatment modulated the status of tumour-associated macrophages (TAMs) from immunosuppressive to activated nature. The activated TAMs produced high levels of IL-12 along with low levels of IL-10 that not only allowed strong Th1 response marked by generation of high levels of IFN-γ but also reduced activation induced T-cell death. CuNG treatment of PBMC from chemo and/or radiotherapy refractory cancer patients also modulated their cytokine status. Most intriguingly, CuNG-treated TAMs could influence reprogramming of TGF-β producing CD4$\pm$CD25$\pm$T-cells towards IFN-γ producing T-cells. CuNG was found to be effective in immunotherapy of drug-resistant cancers through reprogramming of TAMs that in turn reprogrammed the T-cells and reeducated the T-helper function to elicit proper anti-tumorogenic Th1 response that effectively reduce tumour growth.

Phospho P38 was elevated following CuNG treatment *in vitro* which might cause the elevation of IL-12 and change in T-helper response towards Th1. CuNG also down regulated phospho AKT level which remained elevated in TAMs and helps in IL-10 production.

Synthesis and characterization of another RMA, *viz.*, N-(2-methoxyphenyl)-3-methoxysalicylaldimine was also undertaken and X-ray powder structure and anticancer activity of the compound was determined.

The results of the study showed the potential usefulness of CuNG in immunotherapy of drug resistant cancers through programming of TAMs that in turn programme the T cells and reduces the T helper function to elicit proper anti-tumourogenic Th1 response that effectively reduce tumour growth.

The modulation of the behavior of TAMs by ROS generating metal chelate like CuNG is a significant contribution which may pave the way of developing new drugs for the treatment of cancer irrespective of the drug resistance phenotype.

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Cellular Chemotherapy,
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Publications

1. Mookerjee, A., Mookerjee, J., Majumder, B.S., Chatterjee, S., Panda, G.S., Dutta, P., Pal, S., Mukerjee, P., Efferth, T., Roy, S. and Choudhuri, S.K. A novel copper complex induces ROS generation in doxorubicin resistant Ehrlich ascites carcinoma cells and increases activity of antioxidant enzymes in vital organs *in vivo. BMC Can 6*:267, 2006.
2. Mookerjee, A., Basu, J.M., Dutta, P., Majumder, S., Bhattacharyya, S., Baral, R.N., Das, T., Mukherjee, P., Efferth, T., Raha, S., Sa, G., Biswas, J., Pal, S., Roy, S. and Choudhuri, S.K. Overcoming drug resistant cancer by a newly developed copper chelate through host protective cytokine-mediated apoptosis. *Clin Can Res 12*: 4339, 2006.
3. Choudhuri, S.K., Mookerjee, A., Chatterjee, S. and Biswas, J. A novel approach of overcoming multidrug resistance in cancer through immunomodulation. *Int J Mol Med 20*: S5, 2007.

4. Chattopadhyay, B., Basu, S., Chakraborty, P Choudhuri, S.K., Mukherjee, A. and Mukherjee, M. Synthesis, spectroscopic characterization, X-ray powder structure analysis, DFT study and *in vitro* anticancer activity of N-(2-methoxyphenyl)-3-methoxysalicylaldimine. *J Mol Struc 932*: 90, 2009.
5. Majumer, S., Chatterjee, S., Pal, S., Biswas, J., Efferth, T. and Choudhuri, S.K. The role of copper in drug-resistant murine and human tumours. *Biometals 22*:377, 2009.
6. Chatterjee, S., Mookerjee, A., Basu, J.M., Chakraborty, P., Ganguly, A., Adhikary, A., Mukhopadhyay, D., Ganguli, S., Banerjee, R., Ashraf, M., Biswas, J., Das, P.K., Mitali Chatterjee, S., Das, T. and Choudhuri, K. Chelate modulates tumour associated macrophages to promote anti-tumor response of T cells. *Plos One 4*:1, 2009.

Molecular and biochemical characterization of cortico capsular adhesion proteins in human senile cataractous lens

The study was carried out on 127 patients with cataractous lens to isolate and characterize cartico capsular adhesion (CCA) molecules present in human senile cataractous lens and select a pharmacological base that can dissolve CCA *in vivo* and *in vitro*.

Two extra proteins like βB1 crystallin and aldehyde dehydrogenase1A1 (ALDH1A1) were also identified by Western blotting using monoclonal antibodies. The protein mRNA expression was carried out by RT-PCR in cataractous lenses and lens epithelial cells. The βB1 crystallin was found higher expression in CCA patients and in ALDH1A1 no significant differences was noticed. The βB1 crystallin has a greater association with CCA formation. Hence these proteins undergo truncation by proteolytic enzymes. MMP-9 activity was measured in lens epithelial cells (LECs) which are more active and maintain lens transparency. MMP-9 activity was significantly higher in CCA lens epithelial cells as compared to non CCA LECs. The interactions of crystalline form higher molecular weight proteins which don't leak out and are accumulated at the periphery of the lens. These accumulation processes take a long time and number of truncated proteins are gathered in the process. These accumulation processes form a strong adhesion between the lens cortex and capsule. During phacoemulsification, this adhesion acts as a hindrance to both surgeons and patients.

Based on these observations, efforts were made to find a solvent system that can weaken or break this adhesion. The only way in which this adhesion can be removed is by using an irrigating solution during cataract surgery. The most commonly used irrigating solution is the balanced slat solution (BSS). This solution has been modified by the addition of glutathione. The glutathione helps to scavenge free radicals and protect the corneal endothelial cells. It was found that concentration levels of more than 0.4 mM/L GSSG and 0.5 mM/L GSH are suitable because the free glutathione concentration significantly decreased in CCA samples as compared to non CCA samples. Moreover higher molecular weight βB1 crystallins were also found in all the samples by Westeren blotting using βB1 crystallin monoclonal antibodies which shows that glutathione reacted with βB1 crystallin and formed higher molecular weight protein adducts.

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ICMR NEWS

The following meetings of various technical committees/groups of ICMR were held:

Meetings of Task Forces (TFs)/Projects Review Committees/ Groups (PRCs/PRGs) held during January 2010

PRG on Health Systems Research	January 7-8, 2010
TF on Nanomedicine	January 12, 2010
TF on Immunophenotyping of Haematolymphoid Malignancies	January 20, 2010

Meetings of Expert Groups (EGs)/and other Meetings held in January 2010

EG on Role of Hedgehog –GLI Signaling in Stem Cells, Cancer and Cancer Stem Cells	January 8, 2010
Expert Committee on Research Data Repository and Business Intelligence	January 12, 2009

EG to Plan Strategy for National Debate on Guidelines for Stem Cell Research and Therapy	January 18, 2010
EG on Chronic Kidney Disease	January 21, 2010
EG on Geriatrics	January 27, 2010

Participation of ICMR Scientists in Scientific Events

Dr. Ashwini Kumar, Scientist E and Officer-in-Charge, National Institute of Malaria Research Field Station, Goa, participated in the XLIV Annual US Japan Joint Conference on Adaptive and Innate Immune Responses to Neglected Tropical Diseases, at San Diago (January 9-11, 2010).

Dr. A.K. Mukhopadhyaya, Scientist C, National Institute of Cholera and Enteric Diseases (NICED), Kolkata, participated in the Meeting "To Appraise Quality Control and Quality Assurances on Current Practices in Laboratories, at Pemba and Unguja in Zanzibar (January 11-16, 2010).

Dr. N. Arumachalam, Scientist F, Centre for Research in Medical Entomology, Madurai, participated in the Asiana Partnership on Emerging Infectious Disease Research Partner and Network Meeting, at Kunming (January 12-17, 2010).

Dr. Vrinda V. Khale, Scientist F, National Institute for Research in Reproductive Health, Mumbai, participated in the Conference on Actual Problems of the Modern Physiology and Biophysics, at Tashkent (January 27-28, 2010).

Dr. N.V. Giridharan, Scientist F, National Institute of Nutrition, Hyderabad, participated in the I International Workshop on Abdominal Obesity, at Hongkong (January 28-30, 2010).

Dr. G.B. Nair, Director, NICED, Kolkata, participated in the I Meeting of the Committee of the Project on Introduction of Cholera Vaccine in Bangladesh: ICVB, at Dhaka (January 28-30, 2010).

Dr. Poonam Salotra, Scientist E, Institute of Pathology, New Delhi, participated in the Meeting of Steering Committee of RAPSO DI Project at Tunis (January 28-30, 2010).

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Sd/- J.N. Mathur
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