International Radiation Information Center

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This center was re-established in June 1994 in order to reorganize and expand the Data and Specimens Center for the Atomic Bomb Disaster. It supports each division of the RIRBM and other institutes through its collection and analysis of international radiation information, redistributing the results to relevant bodies on a worldwide basis. The projects for research and service are as follows:

1. Research on collection, arrangement, preservation, and analysis of data and specimens from A-bomb disaster victims.
   (Assessment of A-bomb dose at Hiroshima and Nagasaki, and others.)
2. Collecting information on worldwide radioactive contamination and its analysis.
   (Estimation of radiation doses to residents around Chernobyl and Semipalatinsk, and study of diseases caused by the radiation and underlying mechanisms, examination of the actual conditions of the radiation exposed residents in Semipalatinsk by the questionnaire method.)
3. Promotion of international cooperative studies and organizing international symposia on radiation effects.
4. Public program of radiation information.
5. Medical assistance in radiation emergency medicine.

On September 30th, 1999, the criticality accident occurred at the JCO, a nuclear fuel conversion factory. We brigaded an assistant team immediately, and investigated radiation doses for Tokai-mura and Naka-machi residents. Furthermore, in order to grasp the exact complexion of the accident, a detailed investigation was performed and a report was published with many researchers belonging to other universities or national research institutes. The system for medical assistance in radiation emergency medicine has been prepared.

Dr. Ryungsa Kim is a councilor of the Japanese Society of Chemotherapy, the Japanese Breast Cancer Society and Japanese Society of Thyroid Surgical. Dr. Noriyuki Kawano is an advisor of Hiroshima-Semipalatinsk Project.
1. Enhancement of drug sensitivity by antisense Bcl-2 in gastric and breast cancer cells

Kim, R., Emi, M. *1, Tanabe, K. *1 (*1Dept. Surg. Oncol.)

The overexpression of Bcl-2 is involved in drug-resistance in gastric and breast cancer cells. We investigated whether the down regulation of Bcl-2 by antisense Bcl-2 ODNs increased drug-sensitivity in gastric and breast cancer cell lines. The treatment with antisense Bcl-2 increased the sensitivity to CDDP and TXL in MKN-45 gastric cancer cells. The mechanism(s) by which antisense Bcl-2 increased drug sensitivity will be clarified in terms of the signal transduction pathways in apoptosis. The treatment with antisense Bcl-2 in ZR-75-1, BT-474, MDA-MB-231 breast cancer cells enhanced the drug-sensitivity to ADM and TXL associated with apoptosis. The induction of apoptosis is correlated with down-regulation of Bcl-2, increase in Bax, and decrease in pAkt leading to apoptosis. In a similar fashion, antisense Bcl-2 enhanced chemotherapeutic effect in vivo, which is assessed by nude mice xenografts. These findings suggest that AS Bcl-2 ODNs may be an effective chemosensitizer for gastric and breast cancer.

2. Non-antisense effect of antisense Bcl-2

Kim, R., Emi, M. *1, Matsuura, K. *1, Tanabe, K. *1 (*1Dept. Surg. Oncol.)

It has been suggested that non-antisense effect such as immunostimulatory action of antisense Bcl-2 (AS Bcl-2) through CpG-motif is involved in AS Bcl-2-induced antitumor effect. We investigated whether AS Bcl-2 ODNs induced immunostimulatory action in comparison of the control of methylated AS Bcl-2 ODNs. The treatment with AS Bcl-2 ODNs induced splenomegaly in nude mice and immunocompetent mice. The suspension cells derived from the splenomegaly, which are consistent with pDCs and B cells showed the increased expression of CD80, CD86, and CD83, and CD27 for memory B cells. The serum cytokine, IL-12 was also increased by the treatment with AS Bcl-2, whereas IL-6, IFNs, and TNF-α were not increased. The immunostimulatory effect was abrogated by the CpG-methylated AS Bcl-2 ODNs. These findings suggest that AS Bcl-2 ODNs induced Th1 polarization through the secretion of IL-12, which can be connected with activation of T cell response.

3. Non-apoptotic cell death induced by anticancer drug in gastric and breast cancer cells

Kim, R., Emi, M. *1, Tanabe, K. *1 (*1Dept. Surg. Oncol.)

Anticancer drug-induced cell death is classified with apoptotic and non-apoptotic cell death such as autophagic cell death. We investigated anticancer drug-induced autophagic cell death in nude mice system. Using MKN-45 gastric tumor xenograft, the combination effect with anticancer drug (CDDP, 5-FU, TXL) and a proteasome inhibitor, PS-341 was assessed. The each chemotherapeutic effect of CDDP, 5-FU, and TXL was enhanced by the combination with PS-341, which is associated with increase in apoptosis and autophagic cell death. The mechanism by which anticancer drug and combination treatment enhance autophagic cell death is under investigated.

4. Induction of autophagic cell death in drug-resistant breast cancer cells

Kim, R., Uchida, Y. *1, Emi, M. *1, Tanabe, K. *1 (*1Dept. Surg. Oncol.)

Autophagic cell death may play an important role in exerting anticancer drug-induced cell death in cancer cells. There are some interplay between apoptosis and autophagic cell death in response to death triggers. We investigated whether drug resistant cancer cells can induce autophagic cell death instead of apoptosis. Using multidrug resistant breast cancer cell line, MCF-7/TH1000, which is overexpressed with P-glycoprotein, combination treatment with ADM and verapamil reversed the drug resistance. The increased sensitivity to ADM is associated with induction of autophagic cell death, which is inhibited by 3-MA. The constitutive overexpression of Bcl-2, Bcl-xL, and Beclin 1 in the drug resistant cells was decreased following
the treatment. In contrast, treatment with ADM of MCF-7 induced apoptosis, which was associated with increased expression of apoptosis-related proteins. These findings suggest that even though apoptosis-inducing pathway is abrogated in the drug resistant cells, autophagic cell death pathway can be activated in an independent pathway way from apoptosis.

5. Clinical significance of autophagic cell death after neoadjuvant chemotherapy in breast cancer

Kim, R., Arihiro, K.¹¹, Emi, M.,₂², Tanabe, K.²² (*¹ Hiroshima University Hospital, *² Dept. Surg. Oncol.)

Autophagic cell death may play an important role in tumor response. We investigated clinical significance of autophagic cell death in breast cancer, which was treated with EC or CEP/TXT as neoadjuvant chemotherapy prior surgical treatment. For the detection of autophagic cell death, we generated anti-LC3 antibody for immunohistochemical staining. It seems that the induction of autophagic cell death after neoadjuvant chemotherapy are correlated with the reduction of tumor volume, however, further studies will be required for the assessment in clinical significance. In addition, interplay with apoptosis and autophagic cell death after the chemotherapy will be needed, and immunostimulatory action of autophagic cell death, which is mediated by increased of MHC class II-antigen complex for presentation will be assessed.

6. Mechanisms of immune escape and promotion of tumor immunity following neoadjuvant chemotherapy in breast cancer

Kim, R., Arihiro, K.₂², Emi, M.¹¹, Tanabe, K.¹¹ (*¹ Dept. Surg. Oncol., *² Hiroshima University Hospital)

Several mechanisms of immune privilege such as loss of tumor antigen and HLA, and immune suppressive factors have been reported in solid tumor. Tumor-driven soluble factors such as VEGF induce immature dendritic cells (iDCs) from bone marrow, which are recruited to primary tumor site through chemokines and their receptors. Tumor-associated iDCs (TiDCs) are functionally modulated in tumor site and distributed in secondary lymphoid organs and peripheral tissues. Given that DC play a crucial role in initiating tumor immunity, maturation of TiDCs is required for provoking antitumor immune response. Unfortunately, most of TiDCs are already primed with apoptotic cells, which lead to low level of costimulatory molecules and decreased capacity for antigen presentation. Unlike TiDCs, the newly produced iDCs from CD34+ progenitor cells in bone marrow and the released iDCs compartmentalized within tumor after neoadjuvant chemotherapy may be distinct, which can be activated and matured, and migrated to sentinel lymph node where they present tumor antigens to naive T cells. The production of massive cell death and tumor antigens may facilitate the release of iDCs and maturation in the presence of inflammatory cytokines such as IL-1 and TNF-α. The study focused on these hypotheses is under progress.

7. Enhancement of therapeutic effect in combination treatment with imatinib and anticancer drug in gastric and breast carcinoma

Kim, R., Emi, M.¹¹, Tanabe, K.¹¹ (*¹ Dept. Surg. Oncol.)

Imatinib (STI571, Gleevec) is a novel molecular targeting agent, which inhibits the activity of c-kit and PDGFR in gastrointestinal stromal tumors, as well as to inhibit the tyrosine kinase activity of Bcr-Abl in CML and ALL. We conducted preclinical evaluation of therapeutic effect of imatinib and potential synergism in combination with imatinib and anticancer drug in gastric carcinoma cells, in terms of growth inhibition by targeting PDGF/PDGFR signaling pathway. MKN-45 gastric carcinoma cells were employed. Treatment with anticancer drug alone, treatment with imatinib alone, and combination treatment with imatinib and anticancer drug were done, respectively. Imatinib was administered intraperitoneally at 50 mg/kg for consecutive 28 days, whereas anticancer drug was administered at LD₅₀/₃ four times in qwk. Antitumor effect was evaluated by NCI protocol. Apoptosis and gene expression was assessed by TUNEL assay, Western blotting, and immunohistochemical staining, respectively. The treatment with imatinib of MKN-45 gastric
carcinoma xenograft showed antitumor effect to a greater extent than the treatment with 5-fluorouracil (5-FU) or docetaxel (TXT) alone. Further, antitumor effect was significantly enhanced in the combination treatment with imatinib and 5-FU or TXT, compared to the treatment with imatinib, 5-FU or TXT alone. Enhanced antitumor effect in combination treatment with imatinib and anticancer drug was associated with both induction of apoptosis and inhibition of tumor angiogenesis, which was explained with the decreased expression in pAkt, and PDGFR-β, and decreased phosphorylation of PDGFR-β. In the way of similar mechanism, combination treatment with STI571 enhanced the chemotherapeutic effect of TXL and TXT. These results suggest that combination treatment with imatinib and anticancer drugs such as 5-FU and TXT may a new strategy for enhancing therapeutic effect in the treatment gastric carcinoma, due to targeting inhibition of PDGFR-mediated paracrine tumor growth.

8. Rationale for endocrine-chemotherapy in breast cancer

Kim, R., Emi, M. *1, Tanabe, K. *1 ( *Dept. Surg. Oncol.)

The therapeutic efficacy of endocrine-chemotherapy has been revealed in receptor positive and node positive breast cancer patients. However, the survival benefit of endocrine-chemotherapy in these patients seems too small compared to receptor negative patients with node positive. Based on these findings, we are planning to investigate whether the treatment with TAM can decrease the therapeutic effect of anticancer drugs in receptor positive patients with breast cancer. Using in vitro model, the effect of introduction of bcl-2 gene into breast cancer cells will be assessed. Further, whether the treatment with TAM can induce the expression of Bcl-2 associated with drug resistance will also be tested. From these findings, the optimal schedule for endocrine-chemotherapy will be evaluated for adjuvant setting in receptor positive and node positive patients with breast cancer. Treatment with estradiol in MCF-7 increased expression of Bcl-2, resulted in the decrease of sensitivity to anticancer drugs.

9. Potential role of HER-2 in primary breast tumor in bone metastasis

Kim, R., Arihiro, K. *1, Emi, M. *2, Tanabe, K. *2 ( *1Hiroshima University Hospital., *2Dept. Surg. Oncol.)

HER-2 is an important prognostic factor in breast cancer, which plays a critical role in tumor progression and metastasis. The overexpression of HER-2 is observed in 20-30% with micrometastasis in bone marrow of breast cancer. Given that the HER-2-overexpressing breast cancer cells might play a role in promoting bone metastasis through the clonal selection in primary site, the aim of this study is to explore the potential functional role of HER-2 of primary breast tumor in bone metastasis, and how HER-2 is involved in the formation of bone metastasis. Forty eight primary breast tumors with bone metastasis including simultaneous or non-simultaneous metastasis were employed. The expression of hormone receptors, metastasis and growth factor-related proteins such as c-Met, VEGF, MTA-1, Akt, and CXCR4 was assessed by immunohistochemical staining. Of 48 breast tumors, HER-2-positive tumors were observed in 11 tumors (22.9%), whereas HER-2-negative tumors were observed in 37 tumors. There was no significant difference in HER-2 status and clinicopathologic factors in two groups. Although the positive expression of estrogen receptor and progesterone receptor was observed in 2 and 3 cases of 11 HER-2-positive tumors, respectively, there was no extranuclear expression of HRs in these tumors. In the metastasis-related proteins such as c-Met, VEGF, and MTA-1, which are activated by HER-2, only few and some focal expression of these proteins was observed. In contrast, the increased level of pAkt was observed in 9 cases (81.8%) of 11 tumors, and the increased expression of CXCR4 was observed in 6 cases (54.5%) of 11 tumors. The frequency of the increased levels of pAkt and CXCR4 was not significant compared to HER-2-negative tumors. The increased levels of pAkt and CXCR4 are induced by HER-2-dependent and -independent manner, and the activation of HER-2/CXCR4/Akt-signaling pathway in primary tumor may contribute to the formation of bone metastasis in breast cancer.
10. Molecular targeting therapy for anaplastic thyroid cancer

Kim, R., Emi, M.\textsuperscript{*1}, Tanabe, K.\textsuperscript{*1} (\textsuperscript{*1}Dept. Surg. Oncol.)

Anaplastic thyroid cancer is poor prognosis because intensive therapy including surgery, radiation, and chemotherapy is mostly failed. To assess molecular targeting therapy for enhancement of chemotherapeutic effect, we performed a preclinical evaluation of STI571 for inhibition of PDGF/PDGF\textsubscript{R}-signaling pathway in tumor growth. Using 8503C anaplastic thyroid cancer cells, the combination effect of STI571 with anticancer drug (TXL, 5-FU) was evaluated in vitro and in vivo. Although enhancement of drug sensitivity was not observed in vitro, combination effect with STI571 and TXL or 5-FU was observed, which was reached to significant difference compared to drug alone. The enhanced antitumor effect was associated with increase in apoptotic cell death and inhibition of tumor angiogenesis. Further evaluation will be required for targeting PDGF/PDGF\textsubscript{R}-signaling pathway in anaplastic thyroid cancer.

11. Research on human suffering effects of nuclear tests at Semipalatinsk, Kazakhstan: on the basis of questionnaire surveys

Kawano, N., Hirabayashi, K.\textsuperscript{*1}, Matsuo, M.\textsuperscript{*2}, Taooka, Y.\textsuperscript{*3}, Hiraoka, T., Ohtaki, M.\textsuperscript{*1}, Hoshi, M.\textsuperscript{*1} (\textsuperscript{*1}Dept. Rad. Bio., \textsuperscript{*2}Res. Inst. Peace Science, \textsuperscript{*3}Grad. Sch. Biomed. Sci., \textsuperscript{*4}Dept. Environ. Biomet.)

Our research project of the Research Institute for Radiation Biology and Medicine, Hiroshima University, started in 2002 to conduct field research to explore the realities of the radiation exposed population in Semipalatinsk area by means of a questionnaire survey. The survey questions were prepared based on past surveys conducted by Hidankyo (Japan Confederation of A- and H- Bomb Sufferers Organizations), the former Ministry of Health and Welfare, and the municipality of Hiroshima and Nagasaki. We posed 20 questions to examine the experiences of the nuclear tests, health conditions, and exposure path. We also included an open-ended question asking the respondents to write their experiences and feelings concerning the nuclear tests. Personal interviews have been also made. In the surveys made in 2002 and 2004, we collected 706 respondents and 468 testimonies from the residents near the Semipalatinsk Nuclear Test Site. We have tried to identify their experiences of the nuclear tests and their health conditions on the basis of the answers to the questionnaire surveys and testimonies. The data in Semipalatinsk were compared with those of similar surveys in Hiroshima and Nagasaki. We clarified their direct experiences of the nuclear tests, their present health conditions, mental diseases and so on. The results published in the journals including Journal of Radiation Research. We continue this research project in the next year.

12. Content analysis and database of testimonies in Semipalatinsk

Kawano, N., Hirabayashi, K.\textsuperscript{*1}, Matsuo, M.\textsuperscript{*2}, Hiraoka, T., Satoh, K.\textsuperscript{*3}, Ohtaki, M.\textsuperscript{*1} (\textsuperscript{*1}Dept. Rad. Bio., \textsuperscript{*2}Inst. Peace Science, \textsuperscript{*3}Dept. Environ. Biomet.)

We tried to reconstruct the overall image of nuclear tests and their human effects by using the testimonies which were collected in 2002 and 2003. We statistically processed them, and categorized those words and expressions which occurred most frequently in the testimonies, and obtained some forty categories which represent the experiences, feelings, desires of those affected by radiation. Next, we conducted a principal component analysis of the categories. The result shows: (1) The experiences of the nuclear tests are arranged along the time axis, with direct experiences of the nuclear tests forming one coherent part of the perception and memory, and with other subsequent experiences forming another. (2) Of the latter, we can discern a core of the experiences on human effects such as “disease,” “death,” “family,” “radiation,” and so on. (3) And around this core, we see two different trends: one pointing to the current distress and plight, and the other pointing to future fear and hope.
13. Database of the materials concerning the Hiroshima and Nagasaki atomic bombings and other radiation exposures

Kawano, N., Hiroshima University Library, Hoshi, M.*1, Kamiya, K.*2 (*1Dept. Rad. Bio., *2 Dept. Exp. Oncol.)

We, Hiroshima University Library and the Research Institute for Radiation Biology and Medicine, made the database of the materials concerning the Hiroshima and Nagasaki atomic bombings and other radiation exposures. This research project was supported by Grant-in-Aid for Publication of Scientific Research Results. We opened the following data on the web site (http://www.lib.hiroshima-u.ac.jp/abdb/) :

1. Newspaper articles concerning the Hiroshima and Nagasaki atomic bombings and other radiation exposures.
2. The materials related to A-bombs returned by the AFIP in the U.S.
4. Data of physical materials concerning the Hiroshima and Nagasaki atomic bombings.
5. Data of US nuclear tests and the former USSR nuclear tests.

We continued to open other essential materials concerning A-bombs and other radiation exposure.

14. Database of testimonies by Hibakusha in Hiroshima and Nagasaki


We attempt at making database of testimonies by Hibakusha. These Hibakusha's testimonies are collected by International Radiation Information Center and Institute for Peace Science, Hiroshima University. We continue to input full text of testimonies into a computer. Now we can retrieve any words in the testimonies. We will try to open the database to the public, but we consider the issues of Hibakusha's privacy and the copyright on a book. This is a joint research project with Dept. Environ. Biomet. of RIRBM and Institute for Peace Science, Hiroshima University.

15. Making a catalog of materials concerning the Korean victims of the atomic bombings donated by Takashi Hiraoka, the former mayor of Hiroshima

Kawano, N., Koike, S.*1, Ishida, M.*1, Hiraoka, T (*1Hiroshima University Archives)

We attempt to make the materials and data denoted by the former mayor of Hiroshima, Takashi Hiraoka. The materials are concerned with the Korean victims of the atomic bombings. He is the leading authority on the problems of the Korean victims. He is collecting the huge amount of materials. We expect to better describe the whole picture of the actual conditions of the Korean victims by using the Hiraoka's materials. His materials were approximately 1500 and we almost finished to make the catalog.

16. Oral history of Takashi Hiraoka, the former mayor of Hiroshima

Kawano, N., Koike, S.*1, Ishida, M.*1, Hiraoka, T (*1Hiroshima University Archives)

Mr. Takashi Hiraoka served as mayor of Hiroshima for eight years from 1991. He assumed an important role in the local administration of Hiroshima, “City of Peace.” We expect to be shown the future role of Hiroshima as the city of the world's first atomic bombing through a clear understanding of “the Hiraoka’s Hiroshima Peace Administration.” We had an interview with Mr. Hiraoka sixteen times.
17. **Making a catalog of the materials of Kiyoshi Shimizu**

Kawano, N.

Dr. Kiyoshi Shimizu was the first professor at department of epidemiology and social medicine of the Research Institute for Radiation Biology and Medicine. He was not only the second director of our institute but also one of the founder members. He was the leading authority on the social medical research of A-Bomb survivors. He left the huge amount of the materials concerning *Hibakusha*. We attempt to make a catalog of his materials. His materials are invaluable to our research on A-Bomb survivors.

18. **Making a catalog of the materials of Minoru Yuzaki**

Kawano, N.

Professor Minoru Yuzaki was also prominent researcher of social effects on the victims of the atomic bombings. He left the huge amount of the materials concerning *Hibakusha*. We attempt to make a catalog of his materials. His materials are invaluable to our research on A-Bomb survivors.

19. **Research on the socioeconomic and psychological damages of A-bomb survivors for the past sixty years.**

Kawano, N., Ohtaki, M. *¹*, Satoh, K. *¹*, Tonda, T. *¹* (*¹* Dept. Environ. Biomet.)

In 2005, the Asahi Shimbun conducted the questionnaire survey to explore the total picture of damage from the atomic bombs. The Asahi Shimbun sent the questionnaire to approximately forty thousand survivors of A-bombs. We joined the research project to tally the answers to the questionnaire survey and to explore the result.

We have tried to identify the socioeconomic and mental sufferings of A-bomb survivors on the basis of the answers to the questions concerning these two aspects. For the data analysis, we use logistic multiple linear regression analysis. We clarified the continuous mental impacts and social sufferings such as “discrimination against marriage.” The previous studies pointed out that the A-bomb damage affected every aspect of human life, socioeconomic and psychological, and each aspect mutually and dynamically related. The result of our research also showed the same result with the previous studies.

20. **Myelodysplastic syndrome (MDS) among atomic-bomb survivors and radiation-exposed residents near the Semipalatinsk nuclear test site**


Increased risk of myelodysplastic syndrome (MDS) has been shown in atomic-bomb survivors. We showed that AML1 point mutations were found in half of MDS patients among atomic-bomb survivors in Hiroshima. To clarify the molecular mechanism of radiation-induced MDS, we are performing mutation analysis of genes including transcriptional factors and tumor suppressors in the patients with MDS in atomic bomb survivors and radiation-exposed residents near the Semipalatinsk nuclear test site.

21. **Molecular mechanism of secondary myelodysplastic syndrome (MDS) / acute leukemia (AML)**

Harada, Y., Harada, H. *¹*, Ding, Y. *¹*, Kimura, A. *¹* (*¹* Dept. Hematol. Oncol.)

Secondary MDS or AML arise after chemotherapy or radiation exposure for other malignancies. We and others have shown that high frequency of AML1 point mutation or AML1-related translocation was observed in patients with secondary
MDS or AML. To clarify the molecular mechanism of secondary MDS or AML, we are trying to detect AML1 gene alteration induced by radiation or anti-cancer drug in human hematopoietic stem cells purified from cord blood cells.

22. Genetic pathway in molecular pathogenesis of myelodysplastic syndrome (MDS) with AML1 point mutations

Harada, Y., Harada, H., Ding, Y., Imagawa, J., Kyo, T., Kimura, A. (*Dept. Hematol. Oncol., **Hiroshima Asa City Hospital, ***Hiroshima Red Cross Hospital and Atomic-bomb Survivors Hospital)

AML1 mutations have been reported to be frequent in myelodysplastic syndrome (MDS) patients, especially those with refractory anemia with excess blast (RAEB), RAEB in transformation (RAEBt), and AML following MDS (defined these categories as MDS/AML). Although AML1 mutations are suspected to play a pivotal role for developing MDS/AML, acquisition of additional genetic alterations is necessary. We analyzed gene alterations in patients with AML1 mutations, comparing with those without AML1 mutation, and detected specific gene alterations in AML1-mutated MDS/AML patients. Now we are inducing both mutants of these genes and AML1 mutants into hematopoietic stem cells, and analyzing biological changes of these cells. Furthermore, we are going to inject these cells into mice and check development of MDS. Using this approach, we are able to clarify the molecular pathogenesis of MDS.

I. Original papers


20. Takeichi, N.¹, Hoshi, M.¹, Iida, S.¹, Tanaka, K.², Harada, Y., Zhumadilov, Z.², Chaizhunusova, N.¹, Apsalikov, K.N.¹, Nosy, Y.¹, Inaba, T.¹, Tanaka, K.¹, Endo, S.¹ (¹Dept. Radiat. Biophys., ²Institute for Environmental Sciences, ³Semipalatinsk State Medical Academy, ⁴Kazakh Research Institute for Radiation Medicine and Ecology, ⁵Saiseikai Hiroshima Hospital, ⁶Dept. Molec. Oncol.): Nuclear abnormalities in aspirated thyroid cells and chromosome aberrations in lymphocytes of residents near the Semipalatinsk nuclear test site. J. Radiat. Res. 47, Suppl. A171-A177, 2006. (I)


II. Presentations


26. Kawano, N., Remarkable experiences for Hibakusha from the nuclear tests at the Semipalatinsk nuclear test site. 3rd Dosimetry workshop on the Semipalatinsk nuclear test site area with 10th Hiroshima international symposium, Hiroshima, 2005. (C)

27. Kawano, N., Remarkable experiences of the nuclear tests in residents near the Semipalatinsk nuclear test site: analysis based on the questionnaire surveys, 494th Meeting of Hiroshima med. ass., Hiroshima, August 4, 2005. (C)

28. Kawano, N., Psychological and socioeconomic damages of the survivors of the atomic bombings: on the basis of questionnaire survey conducted by Asahi Shinbun, Hiroshima, Japanese joint statistical meeting, September 12, 2005. (C)

29. Kawano, N.: Other victims of radiation exposure than Hiroshima and Nagasaki: effects of radiation exposure on the inhabitants near the Semipalatinsk nuclear test site, Kazakhstan, the Hiroshima University extension course, “Consider a peace in the world from Hiroshima”, Hiroshima university Koujin kaikan, September 14, 2005. (C)


III. Others


2. Harada, Y.: Radiation effects on human---Atomic bomb survivors have increased risks of myelodysplastic syndrome even 50 years after a single radiation exposure. of Fukuyama high school attached to Hiroshima University, Hiroshima, 2005. (in jap.) (C)

Appendix 1

11th Hiroshima International Symposium
-20th anniversary of the Chernobyl accident and related Semipalatinsk problems-

PERIOD: 7 FEBRUARY, 2006
VENUE: HIROSHIMA UNIVERSITY KOJIN KAikan
1-2-3 KASUMI, MINAMI-KU, 734-8551 HIROSHIMA
TELEPHONE:082-257-5098

Sponsorship: Research Institute for Radiation Biology and Medicine, Hiroshima University
Co-sponsorship: Hiroshima University 21st Century COE Program
“Radiation Casualty Medical Research Center”
Hiroshima International Council for Health Care
Organizer: M.Hoshi and M.Ohtaki (Hiroshima University)
Program

10:00  Opening address : F. Suzuki (Hiroshima University)

10:10  Greetings Kazbek Apsalikov (Kazakh Scientific Research Institute for Radiation Medicine and Ecology, Kazakhstan)

Session 1: Chairpersons: M.Ohtaki and N.Whitehead
10:20  Natallia Savva (Deputy Director, Belarusian Research Center for Pediatric Oncology and Hematology, Belarus)  
“Childhood leukemia in Belarus after Chernobyl accident: Period 1990-2004”

11:00  Sergey Ablameyko (General Director, United Institute of Informatics Problems, National Academy of Sciences, Belarus)  
“Image-based information technologies for cancer diagnostics in Belarus”

11:40  Lunch Time

Session 2: Chairpersons: S. Endo and V. Stepanenko
13:10  Neil Whitehead (RIRBM, Hiroshima University)  
“How recoil sputtering affects the weathering of Chernobyl hot particles”

13:50  T. Tonda (Hiroshima University)  
“Statistical analysis of time trend of prefecture-specific cancer mortalities in Japan  
- Preliminary study on analysis of cancer mortality data of Belarus -”

14:30  Coffee break

Session 3: Chairpersons : N.Takeichi(Takeichi clinic), N. Savva (Belarusian Research Center for Pediatric Oncology and Hematology, Belarus) and M.Hoshi (Hiroshima University)
14:40  Sergey Shinkarev (Institute of Biophysics, Russia)  
“Study on whole body and thyroid dose assessment for the general population in Belarus following the Chernobyl accident”

15:20  N.Takeichi (Takeichi clinic)  
“Examination of thyroid in Belarus”

16:00  Coffee break

16:20  Discussion  
M.Hoshi (Hiroshima University), S.Shinkarev (Institute of Biophysics, Russia) and S.Ablameyko (General Director, United Institute of Informatics Problems, National Academy of Sciences, Belarus)

17:00  Closing address  M.Hoshi , Hiroshima University

17:20  Party