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Based on the reorganization of Research Institute for Radiation Biology and Medicine, Department of Developmental Biology and Oncology was newly established and its research activities had started since February 1, 1996. In the second reorganization of the Institute in 2002, the name of our department had changed to the department of Experimental Oncology in order to express the aim of our research more clearly, and belonged to the division of Genome Biology. Major research activities of this department have focused on the study of molecular mechanisms of radiation carcinogenesis including DNA damage response and repair.

The research projects, which were carried out in 2005 and being planned for the following years, are summarized as follows.

1. Research on the molecular mechanisms of radiation carcinogenesis.
2. Studies on the molecular mechanisms of spontaneous and radiation-induced mutations and genetic instability.
3. Studies on the function of translesion DNA synthesis enzyme,REV1, and its role on radiation carcinogenesis.
4. Functional analysis of histone H2AX complex on DNA damage response.
5. Studies on genetic susceptibility to carcinogenesis in radiation-induced mammary cancer of rats.
6. Development of rodent model for carcinogenesis research

1. Reconstitution of DNA replication *in vitro*

Masuda, Y., Yano, M., Kamiya, K.

Aim: In order to study of molecular mechanisms of mutagenesis *in vitro*, the establishment of a reconstitution system for DNA replication is a crucial step. In this study, we focused on a replication system with pol δ, one of major eukaryotic

DNA polymerases, and the auxiliary proteins (RPA, RFC and PCNA).

Results: Pol δ is is heteroheptameric complex (p125, p66, p50, p12), and RFC is a heterpentameric complex (p140, p40, p38, p37, p36) PCNA is a homotrimeric ring-shaped complex (29 kDa for each monomer), and RPA is a heterotrimeric complex (p70, p32, p14) . We established a method for overproduction of those protein factors in *Escherichia coli* cells, and for purification by conventional column chromatography as active protein complexes on *in vitro* DNA replication reaction. We believed that this system accelerate the study of molecular mechanisms of mutagenesis in vitro

2. Identification of native REV1 complex,enzyme of translesion DNA synthesis, and analysis of its function

Tachibana, N., Masuda, Y., Mori, T.*, Kamiya, K. (*Radioisotope Research Center, Nara Medical University)

Purpose: In *Saccharomyces cerevisiae*, it was demonstrated that REV1 possesses a second function in addition to its deoxycytidyltransferase activity. This fact suggests that interaction between REV1 and other proteins might be important. The aim of this study is to identify the specific interaction between REV1 and other proteins in native REV1 complex and detect the second function of REV1.

Method and Result: We had succeeded in developing an anti-REV1 monoclonal antibody which could detect the endogenous hREV1 by immunoprecipitation. We detected the REV1 epitope which was recognized by anti-REV1 antibody. We performed immunoprecipitation-Western blotting analysis with two REV1 antibodies which could identify the different epitope. Furthermore, we examined whether this signal in Western blotting indicated the endogenous hREV1 or not,using the RNA interference (RNAi) technology. Knockdown analysis showed that this signal of REV1 was reduced to approximately 30% of the control. As a result, we succeeded in detecting endogenous hREV1 in human cells by immunoprecipitation-Western blotting analysis. In next experiment, we will detect individual components of REV1 complex by Western blot analysis and mass spectrometry.

3. Study on mutagenic and carcinogenic effects of Rev1,Y-family DNA polymerases, in mice

Kajimura, J., Yoshida, M., Watanabe, H., Honda, H.*1, Masuda, Y.,T., Piao, J. L., Kusunoki, Y.*2, Hayashi, T.*2, Mizuno, K., Kamiya, K. (*1Department of Developmental Biology, *2RERF)

Purpose: Rev1,the member of the Y-family DNA polymerase, has deoxycytidyl transferase activity that incorporates dCMPs on the opposite to an abasic site. The purpose of this study is to determine how the increased function of Rev1 affects the mutagenicity and the susceptibility to tumor induction in *Rev1* transgenic mice.

Methods and Results: We had developed the *Rev1* transgenic mice which were carrying a cDNA of mouse *Rev1* gene under the metallothionein promoter. *Rev1* transgenic mice and wild type mice (C57BL/6N) developed lymphomas. We found that incidence of lymophoma increased in female *Rev1* transgenic mice and that the number of small intestinal tumor increased in male *Rev1* transgenic mice. Cell surface and molecular analyses of lymphoma cells revealed that tumors developed from various differentiation stages of T cells and in some case had point mutations in *Ikaros* or p53 genes, irrespectively of gender or *Rev1* statuses. We also found that the number of small intestinal tumor per mouse increased in *Rev1* homozygous compared with that of wild type and Rev1 hemizygous mice. These results suggest that Rev1 may enhance the susceptibility to tumorigenicity in gene-dose dependent manner in vivo.

4. Identification of amino acid residues responsible for substrate discrimination and enzymatic activity of human REV1 protein

Piao, J.L., Masuda,Y., Kamiya,K.

Purpose: Translesion DNA synthesis (TLS) is one of major post-replication repair pathways, which are a cellular response to the DNA damage. The REV1 protein is a member of the *umuC/dinB/XPV* gene family, and encodes a dCMP

transferase. In this study, we examined roles of amino acid residues for substrate discrimination using purified mutant REV1 proteins.

Results: Structure analysis of humans REV1 protein revealed several amino acid residues conserved in eukaryotes. We produced the mutant REV1 proteins with substitutions in the amino acid residues to examine those activities. Consequently, we identified amino acid residues, which are responsible for substrate discrimination of REV1 protein and TLS activity.

5. The analysis of dynamics of histone H2AX in DNA damage response

Ikura, T.[*1], Tashiro, S.[*2], Kakino, A., Kamiya, K. ([*1]Tohoku Univ., Graduate School of Medicine, [*2]Department of Cellular Biology)

Chromatin organization in nucleosomes and higher order structure plays an important role in such DNA activities as transcription, replication, recombination and repair. Histone proteins are a major component of nucleosome and play a key role in their formation. Core histone proteins are mainly deposited onto and bind statically to chromosomal DNA during S phase whereas certain other histones have been shown to exchange during other portions of the cell cycle. H2AX is phosphorylated after induction of DNA damage (γ -H2AX) and visualized as foci in cell nucleus. However, the binding properties of the histone protein H2AX to DNA in response to double strand breaks (DSB) are unclear, although it has been thought to bind statically. Here we show that H2AX becomes highly mobile after induction of DSBs. We further show that mobilization depends not on phosphorylation but rather on ubiquitination, and that ubiquitination of H2AX is, in turn, regulated by TIP60 histone acetylase which implicates TIP60 in DNA repair. These results suggest that mobilization of H2AX is an early and necessary step in repair of DNA DSB.

Method

1. FRAP analysis in combination with micro-irradiation
2. MS/MS analysis of the purified H2AX complex

Result.

1. GFP-H2AX was highly mobile in microirradiated area.
2. This mobile actin was regulated by ubiquitination.

6. The analysis of the H2AX complex in DNA repair

Kakino, A., Ikura, T.[*1], Tashiro, S.[*2], Kamiya, K. ([*1]Tohoku Univ., Graduate School of Medicine, [*2]Department of Cellular Biology)

Eukaryotic genome is the tightly packed into the chromatin, a hierarchically organized complex of DNA and histone and nonhistone proteins. This packing represents a common obstacle for most of the DNA functions. Thus, chromatin remodeling or histone modification is required for the chromatin repair machinery. However the role of these chromatin modifications in the cellular response to DNA damage remains unclear. Histone H2AX, histone H2A variant, was reported to be phosphorylated after induction of DNA damage and visualized as foci in cell nucleus. Recently, we purified H2AX from HeLa cells stably expressing FLAG/HA tagged H2AX as a protein complex. Our goal is to clarify the mechanism of DNA damaged repair focusing on the purified H2AX complex.

Method

1. Purification of H2AX from HeLa cells after DNA damage
2. MS/MS analysis of the component of the H2AX complex.

Result.

1. Purified the H2AX was included in the protein complex.
2. Some chromatin remodeling factors were identified by MS/MS analysis.

7. Purification and biochemical analysis of human RRM3 protiens

Gu, Y.Q., Masuda,Y., Kamiya,K.

Purpose: *Saccharomyces cerevisiae RRM3* has been identified to have the function of maintaining chromosomal integrity. In its absence, replication fork stalling and breaking of chromosomes increase. In order to investigate *RRM3* gene function of humans, we have started to identify the human *RRM3* gene.

Results: We have cloned the full-length human homologue of *S. cerevisiae RRM3* cDNA, and overexpressed it in *E. coli* cells and later purified the recombinant RRM3 protein. By biochemical assay analysis, we found human RRM3 protein, like its yeast homologue, contains single-stranded DNA dependent ATPase activity. Currently we are investigating the gene function of maintaining chromosomal integrity.

8. Functional analyses of mfleg1 and hFLEG1, which are over expressed in hepatocellular carcinomas

Koike, N., Ikura,T.[*1], Masuda, Y., Sumii, M.[*2], Kamiya, K. ([*1]Tohoku Univ., Graduate School of Medicine, [*2] 1st Dept. Int. Med., Sch. Med.)

Purpose: Using differential display technique, we cloned a gene, *mfleg1*. We also cloned its human homologue, *hFLEG1*. We investigated the functions of these two proteins, which are over expressed in hepatocellular carcinomas (HCCs).

Method and Results: *mfleg1* was about 3900bp in length and encoded a protein of 544 amino acids. *mfleg1* was expressed ubiquitously, but expressed intensely in thymus, ovary and uterus. *mfleg1* was over expressed in 7 of 9 HCCs (78%). Furthermore, HCC cell lines with strong ability of colony formation in soft agar culture were apt to over express *mfleg1* mRNA, suggesting that over expression of *mfleg1* might correlate with anchorage-independent growth. These results indicated that the over expression of mfleg1 might play an important role on mouse hepatocarcinogenesis. Immunohistchemical examinations showed that mfleg1 located in nucleus.

hFLEG1 was about 2500bp in length and encoded a protein of 748 amino acids. hFLEG1 presented 34% identity and 44% similarity to mfleg1. *hFLEG1* was expressed ubiquitously, but expressed intensely in testis and thymus. Because *mfleg1* over expressed in mouse HCCs, we investigated *hFLEG1* levels in human HCCs. hFLEG1 was over expressed in all of 5 HCCs (100%). Furthermore, *hFLEG1* was also over expressed in some malignant cell lines, such as leukemia cell line K-562, colon cancer cell line SW480, prostate cancer cell line PC3 and DU145. These data suggested that over expression of hFLEG1 might play an important role on tumorigenesis in various malignant tumors. Immunohistchemical examinations showed that hFLEG1 located mainly in nucleus. We are further investigating the functions of these two proteins.

9. Functional analysis of CRAD3, modifying factor of mouse hepatocarcinogenesis

Sumii, M.[*1], Yamazaki, T. [*2], Kominami, S. [*], and Kamiya, K. ([*1]Hiroshima Memorial Hospital., [*2]Fac. Integr. Arts Sci.)

Purpose: We identified a mouse novel gene, cis-retinol/androgen short-chain dehydrogenase type 3 (CRAD3), which was over expressed in mouse hepatocellular carcinomas (HCCs), by differential display technique. We investigated the character of CRAD3 and a role of over expressed of CRAD3 on mouse hepatocarcinogenesis.

Result: Like CRAD1 and CRAD2, CRAD3 had oxidative 3α-hydroxysteroid dehydrogenase activities (oxidative 3α-HSD activities), which could oxidize 3α-adiol, inactive androgen, back to dihydrotestosterone, active androgen. Compared with the *Km* values, CRAD3 has more powerful oxidative 3α-HSD activity than CRAD1 or CRAD2 at physiological 3α-adiol level. Moreover, oxidative 3α-HSD activity in mouse HCC was higher than that in normal liver at physiological 3α-adiol level. This increased oxidative 3α-HSD activity in mouse HCC was thought to result from over expressed CRAD3. Dihydrotestosterone is well known to promote hepatocarcinogenesis. Therefore, CRAD3 may promote mouse hepatocarcinogenesis by increasing local dihydrotestosterone level.

10. Studies on genetic predisposition and modifier factors of radiation-induced rat mammary carcinogenesis

Piao, J. L., Kamiya, K.

The growth and development of radiation-induced rat mammary cancers are highly influenced by the host factors such as genetic predisposition/background or hormonal conditions, and environmental factors. In this study, we focus on the analysis of host and environmental factors which modify the growth and development of radiation-induced rat mammary cancers, at the level of molecular, cell, and whole body.

11. Protection of tumor development and prevention of the chemotherapeutic drugs-induced intestinal toxicity by MAK

Kashimoto, N., Watanabe, H., Kamiya, K.

The present study was designed to investigate the effects of MAK in the diet on the development of lung tumors in rats. The numbers, diameters of lung tumors in macroscopical observation and the numbers and sizes of pulmonary tumors in pathological observation in MAK group was significantly decreased as compared with those of lung tumors in the control group. Furthermore MAK was found to decrease the numbers of PCNA strongly positive tumors and increase the number of negative tumors in a dose-dependent manner. It is considered that MAK attenuated the down-regulated cell cycle activity during carcinogenesis. The present results thus indicate that dietary supplementation with MAK exerts chemopreventive effects on lung pulmonary carcinogenesis.

And then, we examined prevention of intestinal toxicity by the chemotherapeutic drugs such as Fluorouracil (5-FU®), combination of Tegafur and Uracil (UFT®), Cisplatin (Randa®), Cyclophosphamide (Endoxan®) and Gefitinib (Iressa®)in mice treated with MAK. MAK significantly increased small intestinal crypt survival. These results suggest that MAK can act as a prevention of small intestinal damage induced by chemotherapic drugs.

Prospective: We are going on the project to mechanisms for prevention of the chemotherapeutic drug -associated toxicity by MAK.

12. Toxicity and carcinogenicity tests of ethanol extract of Hokosshi seeds in rats

Kashimoto, N., Watanabe, H., Kajimura, J., Kamiya, K.

Aim: Ethanol extract of seeds of *Psoralea corylifolia* O.KZE.(Hokosshi) is for food additive use preservation of grilled or fried chicken or pickles in Japan. The main component is the Bactial with antibacterial action. AMES examination (-), mouse small nucleus examination (-), chromosomal aberration test (+) and mouse LD50 (♂ : 6.1g/kg, ♀ : 5.3g/kg) have been reported by toxicity studies. In addition, it is reported that testes atrophy and decrease of body weights are found in rats. We are now going on the project for a 12months repeated dose toxicity and 24month carcinogenicity tests in rats.

The project for a 12months repeated dose toxicity was just finished. As a result, the decrease of body weights was observed at 1% or more in males, and 0.2% or more in females. It was considered that the no observed adverse effect levels (NOAEL) from a Hokosshi extract were 0.2% in males and 0.04% in females. The carcinogenicity test is now going on.

Lists of Contributions

A. Original Papers

1. Kamath-Loeb, A.[*1], Masuda, Y., Hanaoka, F.[*2], Loeb L.A.[*1] ([*1]University of Washington, [*2]RIKEN Discovery Research Institute): The First US-Japan Meeting on Error-Prone DNA Synthesis, Maui, Hawaii, December 20 - 21, 2004. DNA Repair, Jun; 4(6): 740-747, 2005. (G)(I)

2. Kashiwabara, S., Kashimoto, N., Uesaka, T.[*1], Wakabayashi, K.[*2], Kamiya, K., Watanabe, H. ([*1]RIKEN, [*2] National Cancer Center Research Institute) : Tumor Induction by Azoxymethane (AOM) and 2-Amino-1-methyl-6-phenylimidazo [4,5-b] pyridine (PhIP) in F344 Rat Gastric Mucosa Featuring Intestinal Metaplasia Caused by X-irradiation. J. Exp. Clin. Cancer Res., Jun; 24(2): 305-312, 2005. (R)(A)(I)

3. Ohuchi, Y.[*], Myojin, Y.[*], Shimamoto, F.[*], Kashimoto, N., Kamiya, K., Watanabe, H.([*]Prefectural University of Hiroshima): Decrease in size of azoxymethane induced colon carcinoma in F344 rats by 180-day fermented miso. Oncology Reports, Dec; 14(6): 1559-1564, 2005. (A)(I)

4. Kamiya, K.: Carcinogenesis Involves High-frequency Initiation and Suppression of Promotion by Normal Epithelial Cells. Japanese Journal of Cancer Clinics (Jpn. J. Cancer Clin.), Jun; 51(5): 311-318, 2005. (R)(A)

5. Kamiya, K., Masuda, Y.: Mechanism of radiation carcinogenesis. The Journal of The Hiroshima Medical Association, *in press*. (R)(A)(G)

6. Masuda, Y., Piao, JL., Kamiya, K.: Interaction between DNA and human REV1 protein, which is responsible for translesion DNA synthesis.. The Journal of The Hiroshima Medical Association, *in press*. (G)

7. Myojin, Y.[*1], Kashimoto, N., Kashiwabara, S., Kamiya, K., Watanabe, H., Teruya, K.[*2], Shirahata, S. [*2] ([*1]Prefectural University of Hiroshima, [*2]Kyusyu Univ.): The protective effects of fermented milk in small intestinal crypt survival and probability of survival of mice. The Journal of The Hiroshima Medical Association, *in press*.(R)(A)

8. Ohuchi, Y.[*], Myojin, Y.[*], Kashimoto, N., Kajimura, J., Kamiya, K., Watanabe, H. (* Prefectural University of Hiroshima): Prevention by 180-day Fermented Miso of Colon Carcinoma Induced by Azoxymethane in F344 Rats. Miso and Science Technology, *in press*. (A)

B. Meeting Presentations

1. Kashimoto, N.,Kyo, E.[*],Kamiya, K.,Watanabe, H. ([*]Wakunaga Pharmaceutical): Inhibitory effect of a water-soluble extract from cultured medium of Ganoderma lucidum (Rei-shi) mycelia to the induced pulmonary adenocarcinoma by N-nitrosobis(2-hydroxypropyl) amine(BHP) in Wistar Rats. ISCap Symposium , Kyoto, 2005. 5. 20, 21. (Program & Abstracts, p.67, 2005) (A)

2. Kamiya, K.: Molecular Mechanisms of Radiation carcinogenesis. The 46th Annual Meeting of Research Society for Delayed Effects of Atomic Bomb Detonation, Hiroshima, 2005.6.4. (Abstracts, p.19, 2005) (R)(A)(G)

3. Masuda, Y., Piao, J.L., Kamiya, K.: Interaction between DNA and REV1, which is responsible for translesion DNA synthesis. The 46th Annual Meeting of Research Society for Delayed Effects of Atomic Bomb Detonation, Hiroshima, 2005. 6. 4. (Abstracts, p.30, 2005) (R)(G)

4. Myojin, Y.[*1], Kashimoto, N., Kashiwabara, S., Teruya, K.[*2], Shirahata, S.[*2], Kamiya, K., Watanabe, H. ([*1] Fukushima COOP Hospital, [*2] Laboratory of Cellular Regulation Technology, Graduate School of Genetic Resources Technology, Kyusyu University): The protective effects of fermental milk on X-irradiation-induced intestinal and bone marrow damages in B6C3F1 mice. The 46th Annual Meeting of Research Society for Delayed

Effects of Atomic Bomb Detonation, Hiroshima, 2005. 6. 4. (Abstracts, p.32, 2005) (R)(A)

5. Masuda, Y., Piao, J.L., Kamiya, K.: Analysis of REV1-DNA interaction. Heisei 17th Research meeting on mutation and cancer prevention, Seto, 2005. 7. 15, 16. (Abstracts, p.7, 2005) (A)(G)

6. Watanabe, H., Kashimoto, N., Kyo, E.*, Kamiya, K. (*Wakunaga Pharmaceutical): Biological effects of a water soluble extract from cultured media of Ganoderma lucidium. Human Beings and Health Forum of the 21st Century in Changchun, China, Changchun, China, 2005. 8. 20-22. (Proceedings, p.25-26, 2005) (A)

7. Kamiya, K.: Development of New Technology for Radiation Emergency Medicine. The 1st Annual Meeting of Hiroshima Univ.-Nagasaki Univ. Conference, Hiroshima, 2005. 8. 25.(R)(A)(G)

8. Masuda, Y., Piao, JL., Kamiya, K.: Targeting mechanism of REV1 to damaging site of DNA. The 30th Annual Meeting of the Chugoku Radiation Research Society, Hiroshima, 2005. 8. 30.(R)(G)

9. Masuda, Y., Kamiya, K.: Interaction between DNA and REV1, which is responsible for translesion DNA synthesis. Sixty-Fourth Annual Meeting of the Japanese Cancer Association, Sapporo, 2005. 9. 14-16. (Proceedings, p.467, 2005) (R)(G)

10. Masuda, Y.: Analysis of REV1-DNA interaction. Research meeting in National Institute of Genetics: DNA-damage response and ubiquitin-signaling pathway, Mishima, 2005. 9. 21, 22. (Reports, p.16, 2005) (R)(G)

11. Kamiya, K.: Radiation Emergency Medicine Network and the role of Hiroshima University as the Center of Western Japan. The 48th Annual Meeting of The Japan Radiation Research Society / The 1st Asian Congress of Radiation Research, Hiroshima, 2005. 11. 15-17. (Proceedings, p.88, 2005)

12. Masuda, Y., Kamiya, K.: Biochemical analysis of mutagenesis induced by ionizing radiation. The 48th Annual Meeting of The Japan Radiation Research Society / The 1st Asian Congress of Radiation Research, Hiroshima, 2005. 11. 15-17. (Proceedings, p.77, 2005) (R)(G)

13. Gu, Y.Q., Masuda, Y., Kamiya, K.: Cloning of human RRM3 cDNA. The 48th Annual Meeting of The Japan Radiation Research Society / The 1st Asian Congress of Radiation Research, Hiroshima, 2005. 11. 15-17. (Proceedings, p.125, 2005) (R)(G)

14. Masuda, Y., Kamiya, K.: Biochemical analysis of post-replication repair pathway by human REV1 protein. The 22nd Radiation Biology Center International Symposium, Kyoto, 2005. 11. 21, 22. (Abstracts, p.13, 2005) (R)(G)

15. Gu, Y.Q., Masuda, Y., Kamiya, K.: Cloning and Overexpression of a Novel Human Helicase. The 22nd Radiation Biology Center International Symposium, Kyoto, 2005. 11. 21, 22. (Abstracts, p.26, 2005) (R)(G)

16. Masuda. Y., Kamiya. K.: Biochemical analysis of Rev1 function and carcinogenesis in Rev1 transgenic mouse. Heisei 17th annual Meeting of the Research Committee “Analysis of molecular mechanisms of carcinogensis after radiation exposure and it’s application for prevention and treatment” supported by Health and Labour Sience Research Grants of Third Term Comprehensive Control Research for Cancer, Hiroshima, 2005. 12. 6. (R)(A)(G)

17. Masuda. Y., Kamiya. K.: Analysis of DNA binding activity of human REV1 protein, which is responsible for translesion DNA synthesis. The 28th Annual Meeting of the Molecular Biology Society of Japan, Fukuoka, 2005. 12. 7-10. (Abstracts, p.191, 2005) (R)(G)

18. Tachibana, N., Kawamura, T., Masuda, Y., Mori, T.*, Kamiya, K. (* RI, Nara Medical University): Examination of purification method for native REV1. The 28th Annual Meeting of the Molecular Biology Society of Japan, Fukuoka, 2005. 12. 7-10. (Abstracts, p.190, 2005) (R)(G)

19. Kamiya, K., Masuda, Y.: New biochemical function of REV1,translesion DNA synthesis enzyme, and carcinogenesis. Heisei 17th annual Meeting of the Research Committee "Research on molecular mechanisms of human radiation carcinogensis" supported by the Grant-in-Aid for Cancer Research from the Ministry of Health,Labour and Welfare, Hiroshima, 2005. 12. 14. (R)(A)(G)

20. Watanabe, H., Kashimoto, N., Kajimura, J., Kamiya, K.: Miso (Japanese soybean paste) was protective against hypertension compared to same amount of sodium chloride diet in Dahl salt-sensitive rats. The 22rd Annual Meeting of the Japanese Society of Toxicologic Pahology, Kagoshima, 2006. 1. 26, 27. (Proceedings, p.63, 2006) (A)

21. Kashimoto, N., Kyo, E.*1, Watanabe, H., Kamiya, K. (*1Wakunaga Parm. Co.): Inhibition of Lung Tumor Development by MAK in Wistar Rats. 12th Annual Meeting of Jpn. Mibyo Sys. Ass. , Osaka, 2006. 1. 27-28. (A)

22. Tachibana, N., Kawamura, T., Masuda, Y., Mori, T.*, Kamiya, K. (* RI, Nara Medical University): Examination of purification method for native REV1. The Third International Symposiumu of Hiroshima University 21st Century COE Program - Radiation Casualty Medical Research Center - , Hiroshima, 2006. 2. 1, 2. (Abstracts, p.35, 2006) (R)(G)

23. Gu, Y.Q., Masuda, Y., Kamiya, K.: Cloning and Characterization of hRRM3: a human DNA helicase. The Third International Symposiumu of Hiroshima University 21st Century COE Program - Radiation Casualty Medical Research Center - , Hiroshima, 2006. 2. 1-2. (Abstracts, p.33, 2006) (R)(G)

24. Masuda, Y., Kamiya, K.: Function of human REV1 protein on the post-replication repair pathway. The Third International Symposiumu of Hiroshima University 21st Century COE Program - Radiation Casualty Medical Research Center - , Hiroshima, 2006. 2. 1-2. (Abstracts, p.34, 2006) (R)(G)

25. Piao, J.L., Masuda, Y., Kamiya, K.: Over production and purification of the recombinant human REV3 protein. The Third International Symposiumu of Hiroshima University 21st Century COE Program - Radiation Casualty Medical Research Center - , Hiroshima, 2006. 2. 1-2. (Abstracts, p.36, 2006) (R)(G)

26. Kamiya, K.: Network System for Radiation Emergency Medicine and the Role of Hiroshima as the Center of Western Japan. WHO-REMPAN Resional Workshop on Radiation Emergency Medical Preparedness and Response in the Western Pacific Asia, Chiba, 2006. 3. 23, 24.

(R), (A), (G) and (C) are reports on the study using Radiation Experiments, Animal Experiments, Gene Technology Facilities and Studies established at the International Radiation Information Center, respectively (I) indicates printed in the scientific journals listed in Current Contents.