Supplementary Remarks-3: The complete forms of the references of this review article:

[1] Allfrey, V., Faulker, R. M. and Mirsky, A. E.: Acetylation and methylation of histones and their possible role in the regulation of RNA synthesis. Proc. Natl. Acad. Sci. USA, 51, 786-794, 1964.

[2] Brownell, J. E., Zhou, J., Rannali, T., Kobayashi, R., Edmondson, D. G., Roth, S. Y. and Allis, C. D.: Tetrahymena histone acetyltransferase A: a homolog to yeast Gcn5 linking histone acetylation to gene activation. Cell 84, 843-851, 1996.

[3] Ogryzko, V. V., Schiltz, R. L., Russanova, V., Howard, B. H. and Nakatani, Y.: The transcriptional coactivators p300 and CPB are histone acetyltransferases. Cell 87, 953-959, 1996.

[4] Taunton, J., Hassig, C. A. and Schreiber, S. L.: A mammalian histone deacetylase related to the yeast transcriptional regulator Rpd3p. Science 272, 408-411, 1996.

[5] Wolffe, A. P.: Histone deacetylase: a regulator of transcription. Science 272, 371-372, 1996.

[6] Hassig, C. A. and Schreiber, S. L.: Nuclear histone acetylases and deacetylases and transcriptional regulation: HATs off to HDACs. Curr. Opin. Chem. Biol. 1, 300-308, 1997.

[7] Davie, J. R.: Covalent modifications of histones: expression from chromatin templates. Curr. Opin. Genet. Dev. 8, 173-178, 1998.

[8] Luger, K. and Richmond, T. J.: The histone tails of the nucleosome. Curr. Opin. Genet. Dev. 8, 140-146, 1998.

[9] Johnson, C. A. and Turner, B. M.: Histone deacetylases: complex transducers of nuclear signals. Semin. Cell Dev. Biol. 10, 179-188, 1999.

[10] Brown, C. E., Lechner, T., Howe, L. and Workman, J. L.: The many HATs of transcription coactivators. Trends Biochem. Sci. 25, 15-19, 2000.

[11] Cheung, W. L., Briggs, S. D. and Allis, C. D.: Acetylation and chromosomal functions. Curr. Opin. Cell Biol. 12, 326-333, 2000.

[12] Strahl, B. D. and Allis C. D.: The language of covalent histone modifications. Nature 403, 41-45, 2000.

[13] Turner, B. M.: Histone acetylation and an epigenetic code. Bioessays 22, 836-845, 2000.

[14] Jenuwein, T. and Allis, C. D.: Translating the histone code. Science 293, 1074-1080, 2001.

[15] Khochbin, S., Verdel, A., Lemercier, C. and Seigneurin-Berny, D.: Functional significance of histone deacetylase diversity. Curr. Opin. Genet. Dev. 11, 162-166, 2001.

[16] Roth, S. Y. Denu, J. M. and Allis, C. D.: Histone acetyltransferases. Annu. Rev. Biochem. 70, 81-120, 2001.

[17] Carrozza, M. J., Utley, R. T., Workman, J. L. and Cote, J.: The diverse functions of histone acetyltransferase complexes. Trend. Genet. 19, 321-329, 2003.

[18] Yang, X. J. and Seto, E.: Collaborative spirit of histone deacetylases in regulating chromatin structure and gene expression. Curr. Opin. Genet. Dev. 13, 143-153, 2003.

[19] Wang, S., Yan-Neale, Y., Zeremski, M. and Cohen, D.: Transcription regulation by histone deacetylases. Novartis Found Symp. 259, 238-245, 2004. [20] Biel, M., Wascholowski, V. and Giannis, A.: Epigenetics - an epicenter of gene regulation: histones and histone-modifying enzymes. Angew. Chem. Int. Ed. Engl. 44, 3186-3216, 2005.

[21] Margueron, R., Trojer, P. and Reinberg, D.: The key to development: interpreting the histone code? Curr. Opin. Genet. Dev. 15, 163-176, 2005.

[22] Kouzarides, T.: Chromatin modifications and their function. Cell 128, 693-705, 2007.

[23] Allis, C. D., Berger, S. L., Cote, J., Dent, S., Jenuwien, T., Kouzarides, T., Pillus, L., Reinberg, D., Shi, Y., Shiekhattar, R., Shilatifard, A., Workman, J. and Zhang, Y.: New nomenclature for chromatin-modifying enzymes. Cell 131, 633-636, 2007.

[24] Lee, K. K. and Workman, J. L.: Histone acetyltransferase complexes: one size doesn't fit all. Nat. Rev. Mol. Cell Biol. 8, 284-295, 2007.

[25] Goldberg, A. D., Allis, C. D. and Bernstein, B. E.: Epigenetics: a landscape takes shape. Cell 128, 635-638, 2007.

[26] Shahbazian, M. D. and Grunstein, M.: Functions of site-specific histone acetylation and deacetylation. Annu. Rev. Biochem. 76, 75-100, 2007.

[27] Berger, S. L.: The complex language of chromatin regulation during transcription. Nature 447, 407-412, 2007.

[28] Suganuma, T. and Workman, J. L.: Crosstalk among histone modifications. Cell 135, 604-607, 2008.

[29] Kohn, K. W., Aladjem, M. I., Weinstein, J. N. and Pommier, Y.: Chromatin challenges during DNA replication: A systems representation. Mol. Biol. Cell 19, 1-7, 2008.

[30] Selvi, R. B. and Kundu, T. K.: Reversible acetylation of chromatin: implication in regulation of gene expression, disease and therapeutics. Biotech. J. 4, 375-390, 2009.

[31] Javierre, B. M., Hemando, H. and Ballestar, E.: Environmental triggers and epigenetic deregulation in autoimmune disease. Discov. Med. 12, 535-545, 2011.

[32] Bannister, A. J. and Kouzarides, T.: Regulation of chromatin by histone modifications. Cell Res. 21, 381-395, 2011.

[33] Verrier, L., Vandromme, M. and Trouche, D.: Histone demethylases in chromatin cross-talks. Biol. Cell 103, 381-401, 2011.

[34] Butler, J. S., Koutelou, E., Schibler, A. C. and Dent, S. Y.: Histone-modifying enzymes: regulators of developmental decisions and drivers of human disease. Epigenomics 4, 163-177, 2012.

[35] Kooistra, S. M. and Helin, K.: Molecular mechanisms and potential functions of histone deacetylases. Nat. Rev. Mol. Cell. Biol. 13, 297-311, 2012.

[36] Graff, J. and Tsai, L.-H.: Histone acetylation: molecular mnemonics on the chromatin. Nat. Rev. Neurosci. 14, 97-111, 2013.

[37] Chen, T. and Dent, S. Y. R.: Chromatin modifiers and remodellers: regulators of cellular differentiation. Nat. Rev. Genet. 15, 93-106, 2014.

[38] Tee, W.-W. and Reinberg, D.: Chromatin futures and the epigenetic regulation of pluripotency states

in ESCs. Development 141, 2376-2390, 2014.

[39] Morgan, M. A. and Shilatifard, A.: Chromatin signatures of cancer. Genes and Dev. 29, 238-249, 2015.
[40] Staudt, L. M. and Lenardo, M. J.: Immunoglobulin gene transcription. Annu. Rev. Immunol. 9, 373-398, 1991.

[41] Peterson, M. L.: RNA processing and expression of immunoglobulin genes. In Handbook of B and T Lymphocytes (Snow, E. C. ed.) pp321-342, 1994, Academic press, San Diego.

[42] Calame, K. L., Lin, K. I. and Tunyaplin, C.: Regulatory mechanisms that determine the development and function of plasma cells. Annu. Rev. Immunol. 21, 205-230, 2003.

[43] Busslinger, M.: Transcriptional control of early B cell development. Annu. Rev. Immunol. 22, 55-79, 2004.

[44] Su, I. H. and Tarakhovsky, A.: Epigenetic control of B cell differentiation. Semin. Immunol. 17, 167-172, 2005.

[45] Nutt, S. L. and Kee, B. L.: The transcriptional regulation of B cell lineage commitment. Immunity 26, 715-725, 2007.

[46] Ramirez, J., Lukin, K. and Hagman, J.: From hematopoietic progenitors to B cells: mechanisms of lineage restriction and commitment. Curr. Opin. Immunol. 22, 177-184, 2010.

[47] Funahashi, J., Okufuji, T., Ohuchi, H., Noji, S., Tanaka, H. and Nakamura, H.: Role of Pax-5 in the regulation of a mid-hindbrain organizer's activity. Dev. Growth Differ. 41, 59-72, 1999.

[48] Stevens, S., Ong, J., Kim, U., Eckhardt, L. A. and Roeder, R. G.: Role of OCA-B in 3'-IgH enhancer function. J. Immunol. 164, 5306-5312, 2000.

[49] Hannon, G. J.: RNA interference. Nature 418, 244-251, 2002.

[50] Shapiro-Shelef, M., Lin, K. I., McHeyzer-Williams, L. J., Liao, J., McHeyzer-Williams, M. G. and Calame, K.: Blimp-1 is required for the formation of immunoglobulin secreting plasma cells and pre-plasma memory B cells. Immunity 19, 607-620, 2003.

[51] Savitsky, D. and Calame, K.: B-1 B lymphocytes require Blimp-1 for immunoglobulin secretion. J. Exp. Med. 203, 2305-2314, 2006.

[52] Nera, K.-P., Kohonen, P., Narvi, E., Peippo, A., Mustonen, L., Terho, P., Koskele, K., Buerstedde, J.-M. and Lassila, O.: Loss of Pax5 promotes plasma cell differentiation. Immunity 24, 283-293, 2006.

[53] Magari, M., Aya, T., Ikeda, M., Todo, K., Kanayama, N. and Ohmori, H.: Enhancement of antibody production from a chicken B cell line DT40 by reducing Pax5 expression. J. Biosci. Bioeng. 107, 206-209, 2009.

[54] Toman, I., Loree, J., Klimowicz, A. C., Bahlis, N., Lai, R., Belch, A., Pilarski, L. and Reiman, T.: Expression and prognostic significance of Oct2 and Bob1 in multiple myeloma: implications for targeted therapeutics. Leuk. Lymphoma. 52, 659-667, 2011.

[55] Fujita, T. and Fujii, H.: Species-specific 5'-genomic structure and multiple transcription start sites in the chicken Pax5 gene. Gene 477, 24-31, 2011.

[56] John, L. B. and Ward, A. C.: The Ikaros gene family: transcriptional regulators of hematopoiesis and immunity. Mol. Immunol. 48, 1272-1278, 2011.

[57] Takami, Y., Kikuchi, H. and Nakayama, T.: Chicken histone deacetylase-2 controls the amount of the IgM H-chain at the steps of both transcription of its gene and alternative processing of its pre-mRNA in the DT40 cell line. J. Biol. Chem. 274, 23977-23990, 1999.

[58] Takami, Y. and Nakayama, T.: N-terminal region, C-terminal region, nuclear export signal and deacetylation activity of histone deacetylase-3 are essential for the viability of the DT40 chicken B cell line. J. Biol. Chem. 275, 16191-16201, 2000.

[59] Nakayama, T. and Takami, Y.: Participation of histones and histone-modifying enzymes in cell functions through alterations in chromatin structure. J. Biochem. 129, 491-499, 2001.

[60] Takechi, S., Adachi, M. and Nakayama, T.: Chicken HDAC2 down-regulates IgM light chain gene promoter activity. Biochem. Biophys. Res. Commun. 299, 263-267, 2002.

[61] Kikuchi, H., Takami, Y. and Nakayama, T.: GCN5: a supervisor in all-inclusive control of vertebrate cell cycle progression through transcription regulation of various cell cycle-related genes. Gene 347, 83-97, 2005.

[62] Kikuchi, H., Barman, H. K., Nakayama, M., Takami, Y. and Nakayama, T.: Participation of histones, histone modifying enzymes and histone chaperones in vertebrate cell functions. Reviews and Protocols in DT40 Research, Springer-Verlag, Berlin, pp225-243, 2006.

[63] Barman, H. K., Takami, Y., Ono, T., Nishijima, H., Sanematsu, F., Shibahara, K. and Nakayama, T.: Histone acetyltransferase 1 is dispensable for replication-coupled chromatin assembly but contributes to recover DNA damages created following replication blockage in vertebrate cells. Biochem. Biophys. Res. Commun. 345, 1547-1557, 2006.

[64] Nakayama, M., Suzuki, H., Yamamoto-Nagamatsu, N., Barman, H. K., Kikuchi, H., Takami, Y., Toyonaga, K., Yamashita, K. and Nakayama, T.: HDAC2 controls IgM H and L-chain gene expressions via EBF1, Pax5, Ikaros, Aiolos and E2A gene expressions. Genes Cells 12, 359-373, 2007.

[65] Kikuchi, H. and Nakayama, T.: GCN5 and BCR signaling collaborate to induce pre-mature B cell apoptosis through depletion of ICAD and IAP2 and activation of caspase activities. Gene 419, 48-55, 2008.
[66] Barman, H. K., Takami, Y., Nishijima, H., Shibahara, K., Sanematsu, F. and Nakayama, T.: Histone acetyltransferase-1 regulates integrity of cytosolic histone H3-H4 containing complex. Biochem. Biophys. Res. Commun. 373, 624-630, 2008.

[67] Kikuchi, H., Barman, H. K., Nakayama, M., Takami, Y. and Nakayama, T.: Studies on epigenetic control of B cell functions using the DT40 cell line. Advances in Genetics Research 2, Urbano K. V. (Ed.), Nova Science Publishers, Inc. NY, pp153-166, 2010.

[68] Kikuchi, H., Kuribayashi, F., Takami, Y., Imajoh-Ohmi, S. and Nakayama, T.: GCN5 regulates the activation of PI3K/Akt survival pathway in B cells exposed to oxidative stress via controlling gene expressions of Syk and Btk. Biochem. Biophys. Res. Commun. 405, 657-661, 2011.

[69] Kikuchi, H., Kuribayashi, F., Kiwaki, N., Takami, Y. and Nakayama, T.: GCN5 regulates the superoxide-generating system in leukocytes via controlling gp91-phox gene expression. J. Immunol. 186, 3015-3022, 2011.

[70] Kikuchi, H., Kuribayashi, F., Imajoh-Ohmi, S., Nishitoh, Y., Takami, Y. and Nakayama, T.: GCN5 protects vertebrate cells against UV-irradiation via controlling gene expression of DNA polymerase η. J. Biol. Chem. 287, 39842-39849, 2012.

[71] Kikuchi, H., Nakayama, M., Kuribayashi, F., Imajoh-Ohmi, S., Nishitoh, H., Takami, Y. and Nakayama T.: GCN5 is essential for IRF-4 gene expression followed by transcriptional activation of Blimp-1 in immature B cells. J. Leukoc. Biol. 95, 399-404, 2014.

[72] Kikuchi, H., Nakayama, M., Kuribayashi, F., Imajoh-Ohmi, S., Nishitoh, H., Takami, Y. and NakayamaT.: GCN5 is involved in regulation of immunoglobulin heavy chain gene expression in immature B cells.Gene 544, 19-24, 2014.

[73] Escaffit, F., Vaute, O., Chevillard-Briet, M., Segui, B., Takami, Y., Nakayama, T. and Trouche, D.: Cleavage and cytoplasmic relocalization of histone deacetylase 3 are important for apoptosis progression. Mol. Cell. Biol. 27, 554-567, 2007.

[74] Baba, T. W., Giroir, B. P. and Humphries, E. H.: Cell lines derived from avian lymphomas exhibit two distinct phenotypes. Virology 144, 139-151, 1985.

[75] Buerstedde, J.-M. and Takeda, S.: Increased ratio of targeted to random integration after transfection of chicken B cell lines. Cell 67, 179-188, 1991.

[76] Takeda, S., Masteller, E. L., Thompson, C. B. and Buerstedde, J.-M.: RAG-2 expression is not essential for chicken immunoglobulin gene conversion. Proc. Natl. Acad. Sci. USA 89, 4023-4027, 1992.

[77] Takami, Y., Takeda, S. and Nakayama, T.: Targeted disruption of an H3-IV/H3-V gene pair causes increased expression of the remaining H3 genes in the chicken DT40 cell line. J. Mol. Biol. 250, 420-433, 1995.

[78] Seguchi, K., Takami, Y. and Nakayama, T.: Targeted disruption of 01H1 encoding a particular H1 histone variant causes changes in protein patterns in the DT40 chicken B cell line. J. Mol. Biol. 254, 869-880, 1995.

[79] Takami, Y., Takeda, S. and Nakayama, T.: Targeted disruption of H2B-V encoding a particular H2B histone variant causes changes in protein patterns on two-dimensional polyacrylamide gel electrophoresis in the DT40 chicken B cell line. J. Biol. Chem. 270, 30664-30670, 1995.

[80] Takami, Y., Takeda, S. and Nakayama, T.: An approximately half set of histone genes is enough for cell proliferation and a lack of several histone variants causes protein pattern changes in the DT40 chicken B cell line. J. Mol. Biol. 265, 394-408, 1997.

[81] Takami, Y. and Nakayama, T.: One allele of the major histone gene cluster is enough for cell proliferation of the DT40 chicken B cell line. Biochim. Biophys. Acta 1354, 105-115, 1997.

[82] Takami, Y. and Nakayama, T.: A single copy of linker H1 genes is enough for proliferation of the DT40

chicken B cell line, and linker H1 variants participate in regulation of gene expression. Genes Cells 2, 711-723, 1997.

[83] Sanematsu, F., Takami, Y., Barman, H. K., Fukagawa, T., Ono, T., Shibahara, K. and Nakayama, T.: Asf1 is required for viability and chromatin assembly during DNA replication in vertebrate cells. J. Biol. Chem. 281, 13817-13827, 2006.

[84] Takami, Y., Ono, T., Fukagawa, T., Shibahara, K. and Nakayama, T.: Essential role of CAF-1-mediated rapid nucleosome assembly for DNA replication and cell division in vertebrate cells. Mol. Biol. Cell 18, 129-141, 2007.

[85] Toyonaga, K., Kikuchi, H., Yamashita, K., Nakayama, M., Chijiiwa, K. and Nakayama, T.: E2A participates in a fine control of pre-mature B cell apoptosis mediated by B cell receptor signaling via transcription regulations of survivin, IAP2 and caspase-8 genes. FEBS J. 276/5, 1418-1428, 2009.

[86] Kikuchi, H., Yamashita, K., Nakayama, M., Toyonaga, K., Tsuneyoshi, I., Takasaki, M. and Nakayama, T.: Lacking of Aiolos accelerates pre-mature B cell apoptosis mediated by BCR signaling through elevation in cytochrome c release. BBA-Molecular Cell Research 1793, 1304-1314, 2009.

[87] Kikuchi, H., Nakayama, M., Takami, Y., Kuribayashi, F. and Nakayama, T.: Possible involvement of Helios in controlling the immature B cell functions via transcriptional regulation of protein kinase Cs. Results Immunol. 1, 88-94, 2011.

[88] Kikuchi, H., Nakayama, M., Takami, Y., Kuribayashi, F. and Nakayama, T.: EBF1 acts as a powerful repressor of Blimp-1 gene expression in immature B cells. Biochem. Biophys. Res. Commun. 422, 780-785, 2012.

[89] Kikuchi, H., Nakayama, M., Kuribayashi, F., Imajoh-Ohmi, S., Nishitoh, H., Takami, Y. and Nakayama, T.: Protein kinase Cθ gene expression is oppositely regulated by EBF1 and GCN5 in immature B cells. FEBS Lett. 588, 1739-1742, 2014.

[90] Fukagawa, T., Mikami, Y., Nishihara, A., Regnier, V., Haraguchi, T., Hiraoka, Y., Sugata, N., Todokodo, K., Brown, W. and Ikemura, T.: CENP-P, a constitutive centromere component, is required for centromere targeting of CENP-C in vertebrate cells. EMBO J. 20, 4603-4617, 2001.

[91] Chang, H. and Delany, M.-E.: Karyotype stability of the DT40 chicken B cell line: macrochromosome variation and cytogenetic mosaicism. Chromosome Res. 12, 299-307, 2004.

[92] Nakayama, T. and Nakayama, M.: Chromatin Conformation Change Code (4C) theory on Gain of Unprogrammed and New Cell Functions by Means of Irreversible Creation of Chromatin Structure Plasticity with Epigenetic Modifications through Various Generations. pp.1-303, 2018. The 2nd Edition of the retirement commemorative monograph (Monograph) (the 1st Edition of the self-publishing Monograph published in 2015 is available from following new URL: <u>http://hdl.handle.net/10458/5293</u>) is available from following new URL: <u>http://hdl.handle.net/10458/6460</u>.

[93] Nakayama, M. and Nakayama, T.: Generation of Pax5-deficient DT40 mutant cells, Pax5(-), and protein and mRNA levels of IgM H- and L-chains artificially and excessively accumulated in Pax5(-) DT40

mutants are rapidly and dramatically reduced through various generations during continuous cultivation. pp.1-28, 2018. The 2nd Edition of the Paper-2 (the 1st Edition of the Paper-2 published in 2017 is available from following new URL: <u>http://hdl.handle.net/10458/5934</u>) is available from following new URL: <u>http://hdl.handle.net/10458/6503</u>.

The 2nd Edition of the Paper-2 is also the modified version of Chapter-3 of the 2nd Edition of the Monograph (the 1st Edition of the Monograph published in 2015 is available from following new URL: http://hdl.handle.net/10458/5293) as follows: Nakayama, M. and Nakayama, T.: Generation of Pax5-deficient DT40 mutants, Pax5(-), and protein and mRNA levels of IgM H- and L-chains artificially accumulated in Pax5(-) are rapidly and dramatically reduced through various generations during continuous cultivation. In: Chromatin Conformation Change Code (4C) Theory on Gain of Un-programmed and New Cell Functions by Means of Irreversible Creation of Chromatin Structure Plasticity with Epigenetic Modifications through Various Generations, Nakayama, T. and Nakayama, M. (Eds.), pp.45-71, 2018. The 2nd Edition of the Monograph is available from following new URL: http://hdl.handle.net/10458/6460. [94] Takechi, S., Adachi, M. and Nakayama, T.: Cloning and characterization of the chick Oct binding factor OBF-1. Biochim. Biophysica Acta 1577, 466-470, 2002.

[95] Nakayama, M. and Nakayama, T.: Protein and mRNA levels of IgM H- and L-chains artificially and excessively accumulated in HDAC2-deficient DT40 mutants are dramatically reduced through various generations during continuous cultivation. pp.1-34, 2018. The 2nd Edition of the Paper-1 (the 1st Edition of the Paper-1 published in 2017 is available from following new URL: http://hdl.handle.net/10458/5933) is available from following new URL: http://hdl.handle.net/10458/5293) as follows: Nakayama, M. and Nakayama, T.: Protein and mRNA levels of IgM H- and L-chains artificially accumulated in HDAC2-deficient DT40 mutants are dramatically reduced through various generations during continuous cultivations. In: Chromatin Conformation Change Code (4C) Theory on Gain of Un-programmed and New Cell Functions by Means of Irreversible Creation of Chromatin Structure Plasticity with Epigenetic Modifications through Various Generations, Nakayama, T. and Nakayama, M. (Eds.), pp.11-44, 2018. The 2nd Edition of the Monograph is available from following new URL: http://hdl.handle.net/10458/6460.

[96] Nakayama, M. and Nakayama, T.: IgM H- and L-chains artificially and excessively accumulated in HDAC2(-/-) DT40 mutants are gradually and dramatically reduced in distinct ways in individual mutant clones through various generations during continuous cultivation. pp.1-38, 2018. The 2nd Edition of the Paaper-3 (the 1st Edition of the Paper-3 published in 2017 is available from following new URL: http://hdl.handle.net/10458/5935) is available from following new URL: http://hdl.handle.net/10458/6504. The 2nd Edition of the Paper-3 is also the modified version of Chapter-4 of the 2nd Edition of the Monograph (the 1st Edition of the Monograph published in 2015 is available from following new URL:

http://hdl.handle.net/10458/5293) as follows: Nakayama, M. and Nakayama, T.: IgM H- and L-chains accumulated artificially and excessively in HDAC2(-/-) DT40 mutants are dramatically reduced in distinct ways in individual mutant clones through various generations during continuous cultivations. In: Chromatin Conformation Change Code (4C) Theory on Gain of Un-programmed and New Cell Functions by Means of Irreversible Creation of Chromatin Structure Plasticity with Epigenetic Modifications through Various Generations, Nakayama, T. and Nakayama, M. (Eds.), pp.72-104, 2018. The 2nd Edition of the Monograph is available from following new URL: http://hdl.handle.net/10458/6460.

[97] Nakayama, M. and Nakayama, T.: Fundamental and distinct ways for irreversible creation of chromatin structure plasticity of proximal 5'-upstream regions of Pax5, Aiolos, EBF1 and OBF1 genes with epigenetic modifications for gain of new cell function to exclude accumulated IgM H- and L-chains in individual clones of HDAC2(-/-) DT40 mutants through various generations during continuous cultivation. pp.1-70, 2018. The 2nd Edition of the Paper-4 (the 1st Edition of the Paper-4 published in 2017 is available from following new URL: http://hdl.handle.net/10458/5936) is available from following new URL: http://hdl.handle.net/10458/6505. The 2nd Edition of the Paper-4 is also the modified version of Chapter-5 of the 2nd Edition of the Monograph (the 1st Edition of the Monograph published in 2015 is available from following new URL: http://hdl.handle.net/10458/5293) as follows: Nakayama, M. and Nakayama, T.: A fundamental way for irreversible creation of chromatin structure plasticity with epigenetic modifications for gaining new cell function to exclude IgM H- and L-chains accumulated in HDAC2(-/-) DT40 mutants through various generations during continuous cultivation. In: Chromatin Conformation Change Code (4C) Theory on Gain of Un-programmed and New Cell Functions by Means of Irreversible Creation of Chromatin Structure Plasticity with Epigenetic Modifications through Various Generations, Nakayama, T. and Nakayama, M. (Eds.), pp.105-167, 2018. The 2nd Edition of the Monograph is available from following new URL: http://hdl.handle.net/10458/6460.

[98] Nakayama, T. and Nakayama, M.: Chromatin conformation change code (4C) theory: A bio-system for gaining un-programmed and new cell functions by means of irreversible creation of chromatin structure plasticity with epigenetic modifications through various generations. pp.1-43, 2018. The 2nd Edition of the Paper-5 (the 1st Edition of the Paper-5 published in 2017 is available from following new URL: http://hdl.handle.net/10458/5937) is available from following new URL: http://hdl.handle.net/10458/6506. The 2nd Edition of the Paper-5 is also the modified version of Chapter-6 of the 2nd Edition of the Monograph (the 1st Edition of the Monograph was published in 2015 and is available from following new URL: http://hdl.handle.net/10458/5293) as follows: Nakayama, M. and Nakayama, T.: Chromatin conformation change code (4C) theory: A bio-system for gaining un-programmed and new cell functions by means of irreversible creation of chromatin structure plasticity with epigenetic modifications through various generations. In: Chromatin Conformation Change Code (4C) Theory on Gain of Un-programmed and New Cell Functions by Means of Irreversible Creation of Chromatin Structure Plasticity with Epigenetic Modifications through Various Generations, Nakayama, T. and Nakayama, M. (Eds.), pp.168-

202, 2018. The 2nd Edition of the Monograph is available from following new URL: http://hdl.handle.net/10458/6460.

[99] Nakayama, M. and Nakayama, T.: IgM H- and L-chains accumulated excessively in HDAC2(-/-) DT40 mutants are dramatically reduced in distinct ways in individual mutant clones through various generations during continuous cultivation. Current Topics in Biochemical Research. 18, 11-25, 2017.

The article is available from following new URL and Website: <u>http://hdl.handle.net/10458/6375</u> and <u>http://www.researchtrends.net/tia/title_issue.asp?id=40&in=0&vn=18&type=3</u>.

[100] Nakayama, M. and Nakayama, T.: Irreversible creation of chromatin structure plasticity of proximal 5'-upstream regions of Pax5, Aiolos, EBF1 and OBF1 genes with epigenetic modifications to exclude IgM H- and L-chains accumulated in individual clones of HDAC2(-/-) DT40 mutants through various generations during continuous cultivation. Current Topics in Biochemical Research. 18, 33-56, 2017.

The article is available from following new URL and Website: <u>http://hdl.handle.net/10458/6376</u> and <u>http://www.researchtrends.net/tia/title_issue.asp?id=40&in=0&vn=18&type=3</u>.

[101] Nakayama, T. and Nakayama, M.: Chromatin conformation change code (4C) theory: A bio-system to gain un-programmed and new cell functions by means of irreversible creation of chromatin structure plasticity with epigenetic modifications through various generations. Current Topics in Biochemical Research. 18, 65-86, 2017.

The article is available from following new URL and Website: <u>http://hdl.handle.net/10458/6377</u> and <u>http://www.researchtrends.net/tia/title_issue.asp?id=40&in=0&vn=18&type=3</u>.

[102] Nakayama, T. and Nakayama, M.: An all-inclusive review: Chromatin conformation change code (4C) theory on a bio-system to gain un-programmed and new cell functions by means of irreversible creation of chromatin structure plasticity with epigenetic modifications through various generations in higher eukaryotes. pp.1-75, 2018. The 2nd Edition of the Paper-6 (the 1st Edition of the Paper-6 published in 2017 is available from following new URL: http://hdl.handle.net/10458/5941) is available from following new URL: http://hdl.handle.net/10458/5293) as follows: Nakayama, T. and Nakayama, M.: All-inclusive review and history on the chromatin conformation change code (4C) theory: A bio-system for gaining un-programmed and new cell functions by means of irreversible creation of chromatin structure plasticity with epigenetic modifications through various generations. In: Chromatin Conformation Change Code (4C) Theory on Gain of Un-programmed and New Cell Functions by Means of Irreversible Creation

[103] Nakayama, T. and Nakayama, M.: A comprehensive and detailed review on the chromatin conformation change code (4C) theory: A theory on ways to gain un-programmed and new cell functions

by means of irreversible creation of chromatin structure plasticity with epigenetic modifications through numerous generations in higher eukaryotes. pp.1-80, 2020. The 4th Edition of the Paper-6 (the 1st, 2nd and 3rd Editions published in 2017, 2018 and 2019 are available from following new URLs: http://hdl.handle.net/10458/5941, http://hdl.handle.net/10458/6507 and http://hdl.handle.net/10458/6613) is available from following new URL: http://hdl.handle.net/10458/6902. The 4th Edition of the Paper-6 is also the modified version of Chapter-7 of the 2nd Edition of the Monograph (the 1st Monograph published in 2015 is available from following new URL: http://hdl.handle.net/10458/5293) as follows: Nakayama, T. and Nakayama, M.: All-inclusive review and history on the chromatin conformation change code (4C) theory: A bio-system for gaining un-programmed and new cell functions by means of irreversible creation of chromatin structure plasticity with epigenetic modifications through various generations. In: Chromatin Conformation Change Code (4C) Theory on Gain of Un-programmed and New Cell Functions by Means of Irreversible Creation of Chromatin Structure Plasticity with Epigenetic Modifications through Various Generations, Nakayama, T. and Nakayama, M. (Eds.), pp.203-260, 2018. The 2nd Edition of the Monograph is available from following new URL: http://hdl.handle.net/10458/6460.

[104] Zimmer, C. and Emlen, D. J.: The Japanese Version of Evolution: Making Sense of Life (2017).

[105] Cooper, V. S. and Lenski, R. E.: The population genetics of ecological specialization in evolving Escherichia coli populations. Nature, 407, 736-739, 2000.

[106] Blount, Z. D., Borland, C. Z. and Lenski, R. E.: Historical contingency and the evolution of a key innovation in a population of Escherichia coli. Proc. Natl. Sci. USA, 105, 7899-7906, 2008.

[107] Kawasaki, M.: Temperature adaptation in Drosophila melanogaster. Konchu, 44, 530-536, 1976 (in Japanese).

[108] Rice, T. and Salt, G. W.: Speciation via disruption selection on habitat preference: Experimental evidence. The American Naturalist, 131, 911-917, 1988.

[109] Nono, M., Kishimoto, S., Sato-Carlton, A., Carlton, P. M., Nishida, E. and Uno, M.: Intestine-togermline transmission of epigenetic information intergenerationally ensures systemic stress resistance in C. elegans. Cell Reports 30, 3207-3217, 2020.

[110] Wenzel, D., Palladino, F., and Jedrusik-Bode, M.: Epigenetics in C. elegans: facts and challenges. Genesis 49, 647–661, 2011.

[111] Gonzalez-Aguilera, C., Palladino, F., and Askjaer, P.: C. elegans epigenetic regulation in development and aging. Brief. Funct. Genomics 13, 223–234, 2014.

[112] Jin, C., Li, J., Green, C.D., Yu, X., Tang, X., Han, D., Xian, B., Wang, D., Huang, X., Cao, X., Yan, Z., Hou, L., Liu, J., Shukeir, N., Khaitovich, P., Chen, C. D., Zhang, H., Jenuwein, T. and Han, J-D. J.: Histone demethylase UTX-1 regulates C. elegans life span by targeting the insulin/IGF-1 signaling pathway. Cell Metab. 14, 161–172, 2011.

[113] Siebold, A.P., Banerjee, R., Tie, F., Kiss, D.L., Moskowitz, J., and Harte, P.J.: Polycomb Repressive Complex 2 and Trithorax modulate Drosophila longevity and stress resistance. Proc. Natl. Acad. Sci. USA 107, 169-174, 2010.

[114] van Oosten-Hawle, P., Porter, R.S., and Morimoto, R.I.: Regulation of organismal proteostasis by transcellular chaperone signaling. Cell 153, 1366–1378, 2013.

[115] Tatum, M.C., Ooi, F.K., Chikka, M.R., Chauve, L., Martinez-Velazquez, L.A., Steinbusch, H.W.M., Morimoto, R.I., and Prahlad, V.: Neuronal serotonin release triggers the heat shock response in C. elegans in the absence of temperature increase. Curr. Biol. *25*, 163–174, 2015.

[116] Greer, E.L., Maures, T.J., Hauswirth, A.G., Green, E.M., Leeman, D.S., Maro, G.S., Han, S., Banko, M.R., Gozani, O., and Brunet, A.: Members of the H3K4 trimethylation complex regulate lifespan in a germline-dependent manner in C. elegans. Nature 466, 383–387, 2010.

[117] Dias, B.G., and Ressler, K.J.: Parental olfactory experience influences behavior and neural structure in subsequent generations. Nat. Neurosci. 17, 89–96, 2014.

[118] Kishimoto, S., Uno, M., Okabe, E., Nono, M., and Nishida, E.: Environmental stresses induce transgenerationally inheritable survival advantages via germline-to-soma communication in Caenorhabditis elegans. Nat. Commun. 8, 14031, 2017.

[119] Vaiserman, A.M., Koliada, A.K., and Jirtle, R.L.: Non-genomic transmission of longevity between generations: potential mechanisms and evidence across species. Epigenetics Chromatin 10, 38, 2017.

[120] Rechavi, O., Houri-Zeevi, L., Anava, S., Goh, W.S.S., Kerk, S.Y., Hannon, G.J., and Hobert, O.: Starvation-induced transgenerational inheritance of small RNAs in C. elegans. Cell 158, 277–287, 2014.

[121] Posner, R., Toker, I.A., Antonova, O., Star, E., Anava, S., Azmon, E., Hendricks, M., Bracha, S., Gingold, H., and Rechavi, O.: Neuronal Small RNAs Control Behavior Transgenerationally. Cell 177, 1814–1826.e15, 2019.

[122] Klosin, A., Casas, E., Hidalgo-Carcedo, C., Vavouri, T. and Lehner, B.: Transgenerational transmission of environmental information in C. elegans. Science, 356, 320-323, 2017.

[123] Zenk, F., Loeser, E., Schiavo, R., Kilpert, F., Bogdanovic, O. and Lovino, N.: Germ lineinherited H3K27me3 restricts enhancer function during maternal-to-zygotic transition. Science, 357, 212-216, 2017.

[124] Moore, M. S., Keletsky, R. and Murphy, C. T.: Piwi/PRG-1 argonaute and TGF- β mediate transgenerational learned pathogenic avoidance. Cell, 177, 1827-1841, 2019.

[125] Nakayama, T. and Nakayama, M.: Chromatin conformation change code (4C) theory: A bio-system for gaining un-programmed and new cell functions by means of irreversible creation of chromatin structure plasticity with epigenetic modifications through numerous generations (3rd Edition). pp.1-51, 2021. The 3rd Edition of the Paper-5 (the 1st and 2nd Editions of the Paper-5 published in 2017 and 2018 are available from following new URLs: http://hdl.handle.net/10458/65937 and http://hdl.handle.net/10458/6596) is available from following new URL: http://hdl.handle.net/10458/6596). The 3rd Edition of the Paper-5 is also the modified version of Chapter-6 of the 2nd Edition of the Monograph (the 1st Edition of the Monograph was published in 2015 and is available from following new

URL: <u>http://hdl.handle.net/10458/5293</u>) as follows: Nakayama, M. and Nakayama, T.: Chromatin conformation change code (4C) theory: A bio-system for gaining un-programmed and new cell functions by means of irreversible creation of chromatin structure plasticity with epigenetic modifications through various generations. In: Chromatin Conformation Change Code (4C) Theory on Gain of Un-programmed and New Cell Functions by Means of Irreversible Creation of Chromatin Structure Plasticity with Epigenetic Modifications through Various Generations, Nakayama, T. and Nakayama, M. (Eds.), pp.168-202, 2018. The 2nd Edition of the Monograph is available from following new URL: <u>http://hdl.handle.net/10458/6460</u>.

[126] Nakayama, T. and Nakayama, M.: A comprehensive and detailed review on the chromatin conformation change code (4C) theory: A theory on ways to gain un-programmed and new cell functions by means of irreversible creation of chromatin structure plasticity with epigenetic modifications through numerous generations in higher eukaryotes (5th Edition). pp.1-85, 2021. The 5th Edition of the Paper-6 (the 1st, 2nd, 3rd and 4th Editions published in 2017, 2018, 2019 and 2020 are available from following new URLs: <u>http://hdl.handle.net/10458/5941</u>, <u>http://hdl.handle.net/10458/6613</u> and <u>http://hdl.handle.net/10458/6902</u>) is available from following new URL: http://hdl.handle.net/10458/6902.

The 5th Edition of the Paper-6 is also the modified version of Chapter-7 of the 2nd Edition of the Monograph (the 1st Monograph published in 2015 is available from following new URL: <u>http://hdl.handle.net/10458/5293</u>) as follows: Nakayama, T. and Nakayama, M.: All-inclusive review and history on the chromatin conformation change code (4C) theory: A bio-system for gaining un-programmed and new cell functions by means of irreversible creation of chromatin structure plasticity with epigenetic modifications through various generations. In: Chromatin Conformation Change Code (4C) Theory on Gain of Un-programmed and New Cell Functions by Means of Irreversible Creation of Chromatin Structure Plasticity with Epigenetic Modifications through Various Generations, Nakayama, T. and Nakayama, M. (Eds.), pp.203-260, 2018. The 2nd Edition of the Monograph is available from following new URL: <u>http://hdl.handle.net/10458/64660</u>.