BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors.
Follow this format for each person. **DO NOT EXCEED FIVE PAGES.**

NAME: Joomyeong 'Joo' Kim

eRA COMMONS USER NAME (credential, e.g., agency login): JOOKIM

POSITION TITLE: Professor of Genetics

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Seoul National University, Seoul, South Korea	BS	02/1986	Microbiology
Seoul National University, Seoul, South Korea	MS	02/1988	Microbiology
Louisiana State Univ Medical Center at New Orleans	PhD	05/1995	Molecular Biology
Oak Ridge National Laboratory	Postdoc	1997	Molecular Genetics

A. Personal Statement

Dr. Joomyeong 'Joo' Kim has been trained as a molecular biologist through PhD training and as a mouse geneticist through postdoctoral training. He has also obtained many bioinformatics skills while working as a staff scientist at the Human Genome Center of LLNL. Thus, these disciplines have become main intellectual foundations for his research group at Louisiana State University. As an instructor, he teaches introductory genetics for undergraduates and epigenetics for graduate students. In terms of mentoring, he has trained about 25 undergraduates, the majority of whom have been supported through HHMI intern program. He has been also training 13 PhD students: 12 of them have successfully obtained their PhD degree. His former students have moved to several prominent institutions as a postdoc, and five of his former students have obtained faculty position after their postdoctoral training. He has also worked with 8 postdoctoral students, three of whom have obtained their academic position. More information can be available through the lab website (http://sites01.lsu.edu/faculty/jkim/).

In the past 30 years, his research group has characterized an imprinted domain called the *Peg3* domain. According to the accumulated results, this imprinted domain is controlled through two *cis*-regulatory elements: the Peg3-DMR (Differentially Methylated Region) and the oocyte-specific alternative promoter of *Peg3*. The Peg3-DMR functions as an ICR (Imprinting Control Region) for the *Peg3* domain. The maternal-specific DNA methylation on the Peg3-DMR is thought to be established through U1-driven transcription during oogenesis. Deletion of this alternative promoter results in loss of the oocyte-specific DNA methylation on the Peg3-DMR, causing biallelic expression or 2x dosage of *Peg3*. Detailed analyses of the mutants with 2x *Peg3* derived a set of unusual phenotypes: delayed parturition and defects in placentophagy as well as nest-building behaviors[1]. This set of phenotypes were also detected only from the homozygotes producing the entire litter with 2x *Peg3*, but not from the heterozygotes producing a half of litter with 2x *Peg3*. This suggests the presence of maximum tolerable dosage of *Peg3* for healthy pregnancy and further fetal influence on maternal pregnancy through the gene dosage of the *Peg3* domain. Also, recent comparative genomic and transcriptomic analyses indicated that the oocyte-specific alternative promoter U1 has been derived from an ancient retrotransposon[2]. Thus, his research group is now focusing on two different directions for the *Peg3* domain: one is to characterize the functional aspect and the other is to characterize the evolutionary aspect of the *Peg3* domain.

1. Kim J. (2025) Fetal influence on maternal pregnancy through the *Peg3* imprinted domain. https://doi.org/10.64898/2025.11.28.691217.
2. Kim J. (2025) Ancient retrotransposon-derived promoters for mammalian genomic imprinting. https://doi.org/10.1101/2025.07.14.664778.

B. Positions and Honors

Positions and Employment

1989 -1990	Researcher, Korea Institute of Science and Technology, Seoul, Korea.
1992 -1994	Teaching Assistant, Nursing Biochemistry, LSU Med. Ctr., New Orleans, LA.
1997 - 2004	Biomedical Staff Scientist, Lawrence Livermore National Lab, CA.
2004 - 2011	Associate Professor, Dept. of Biological Sciences, Louisiana State University, LA.
2011 - now	Professor, Dept. of Biological Sciences, LSU, LA.
2011 - 2012	George C. Kent Professor, Dept. of Biological Sciences, LSU, LA.
2012 - now	Russell Thompson Jr. Family Professor, Dept. of Biological Sciences, LSU, LA.

Other Experience and Professional Memberships

2004 - now	Member of Editorial Board for Analytical Biochemistry
2004 - now	Ad hoc reviewer for Hum Mol Genet, Genome Research, Nucleic Acids Res, PLoS Genetics, Genomics, PLoS ONE
2004	NIH, Molecular Genetics Study Section B (MGB)
2006	NIH, Molecular Genetics Study Section B (MGB)
2008	NIH, Fellowship Grant Study Section (GGG)
2009	NIH ARRA RC1 mail reviewer
2011	NIH, Fellowship Grant Study Section (GGG)
2013	NIH, NIEHS RFA Transgenerational effects of environmental exposures
2013	NIH, Genetics of Health and Disease (GHD)
2014	NIH, Fellowship Grant Study Section (GGG)
2015	NIH, Fellowship Grant Study Section (GGG)
2015	NIH, Genetics of Health and Disease (GHD)
2016	NIH, Genetics of Health and Disease (GHD)
2017	NIH, Genetics of Health and Disease (GHD)

C. Contribution to Science

1. In the past 27 years, Dr. Kim's research has focused on characterizing the *Peg3* imprinted domain. His group has identified and characterized 7 imprinted genes within this domain. Also, his group has identified several *cis*-regulatory elements that may be involved in the transcription and imprinting control of this domain. This includes three DMRs (Differentially Methylated Regions), 19 ECRs (Evolutionarily Conserved Regions), and three alternative promoters. One of the DMRs turns out to be an ICR for this domain, and also an oocyte-specific alternative promoter U1 of *Peg3* is shown to be responsible for setting up allele-specific DNA methylation on this ICR.

    a. He H, Kim J. (2014) Regulation and function of the Peg3 imprinted domain. *Genomics Inform* **12(3)**:105-113.
    b. Thiaville MM, Kim H, Frey WD, Kim J. (2013) Identification of an evolutionarily conserved *cis*-regulatory element controlling the Peg3 imprinted domain. *PLoS One* **8(9)**:e75417.
    c. Perera BP, Kim J. (2015) Alternative promoters of Peg3 with maternal specificity. *Sci Rep* **6**:24438.
    d. Kim J, Ye A. (2016). Phylogenetic and epigenetic footprinting of the enhancers of the Peg3 domain. *PLoS One* **11(4)**:e0154216.
    e. Bretz CL, Kim J. (2017) Transcription-driven DNA methylation setting on the mouse Peg3 locus. *Epigenetics* **12(11)**:945-952.

2. Dr Kim's group has also investigated potential protein functions of PEG3. According to the results, PEG3 is a DNA-binding protein controlling a large number of downstream genes. Some of the notable downstream targets include H19 and the two components of the mammalian MSL (Male Sex Lethal) complex, Msl1 and Msl3. Thus, PEG3 may control its downstream genes through the MSL complex involving the H4K16ac modification. PEG3 also interacts with the co-repressor KAP1 through a KRAB-A box, confirming that PEG3 is a transcriptional repressor that may have originated from an ancestral KRAB-containing zinc finger gene.

    a. Kim J, Frey WD, He H, Kim H, Ekram MB, Bakshi A, Faisal M, Perera BPU, Ye A, Teruyama R. (2013) Peg3 mutational effects on reproduction and placenta-specific gene families. *PLoS One* **8(12)**:e85477.
    b. Lee S, Ye A, Kim J. (2015) DNA-binding motif of the imprinted transcription factor PEG3. *PLoS One* **10(12)**:e0145531.
    c. Ye A, He H, Kim J. (2016) PEG3 binds to H19-ICR as a transcriptional repressor. *Epigenetics* **11(12**):889-900.
    d. Ye A, Kim H, Kim J. (2017) PEG3 control on the mammalian MSL complex. *PLoS One* **12(6)**:e0178363.
    e. He H, Ye A, Kim H, Kim J. (2016) PEG3 interacts with KAP1 through KRAB-A. *PLoS One* **11(11)**:e167541.

3. Dr. Kim's earlier research was focused on characterizing potential roles of YY1 in imprinting regulatory mechanisms for the *Peg3* domain. His group discovered that the ICR of the *Peg3* domain contains an unusual tandem array of YY1 binding sites, and subsequently investigated potential roles of YY1 in the *Peg3* domain. His group also expanded this finding to the other imprinted loci, including *Gnas* and *Xist/Tsix*. According to the results, YY1 is not involved in DNA methylation setting, instead controls the transcriptional levels of the *Peg3* domain. While focusing on the *Peg3* domain, his group also characterized the phylogenetic relationship among the three YY1- related genes, YY1, YY2 and Rex1.

    a. Kim J, Kollhoff A, Bergmann A, Stubbs L. (2003) Methylation-sensitive binding of transcription factor YY1 to an insulator sequence within the paternally expressed imprinted gene, Peg3. *Hum Mol Genet* **12(3)**:233-245.
    b. Kim JD, Hinz AK, Bergmann A, Huang JM, Ovcharenko I, Stubbs L, Kim J. (2006) Identification of clustered YY1 binding sites in imprinting control regions. *Genome Res* **16(7)**:901-911.
    c. Kim JD, Kang K, Kim J. (2009) YY1's role in DNA methylation of Peg3 and Xist. *Nucleic Acids Res* **37(17)**:5656-5664.
    d. Perera BP, Teruyama R, Kim J. (2015) Yy1 gene dosage effect and bi-allelic expression of Peg3. *PLoS One* **10(3)**:e0119493.
    e. He H, Ye A, Perera BPU, Kim J. (2017) YY1's role in the Peg3 imprinted domain. *Sci Rep* **7**:6427.

4. As a separate project, Dr Kim's group has been characterizing another zinc finger DNA-binding protein, called *Aebp2* (Adipocyte Enhancer Binding Protein 2). According to the results, this gene is a component of Polycomb Repression Complex 2 (PRC2), yet highly expressed in neural crest cells. His group has published several studies describing the basic features of this gene, including its detailed expression patterns, DNA-binding motifs, and potential roles in neural crest cells as an epigenetic regulator.

    a. Kim H, Kang K, Kim J. (2009) AEBP2 as a potential targeting protein for Polycomb Repression Complex PRC2. *Nucleic Acids Res* **37(9)**:2940-2950.
    b. Kim H, Kang K, Ekram MB, Roh TY, Kim J. (2011) Aebp2 as an epigenetic regulator for neural crest cells. *PLoS One* **6(9)**:e25174.
    c. Kim H, Ekram MB, Bakshi A, Kim J. (2015) AEBP2 as a transcriptional activator and its role in cell migration. *Genomics* **105(2)**:108-115.
    d. Kim H, Bakshi A, Kim J. (2015) Retrotransposon-Derived Promoter of Mammalian Aebp2. *PLoS One* **10(4)**:e0126966.
    e. Kim H, Langohr IM, Faisal M, McNulty M, Thorn C, Kim J. (2018) Ablation of Ezh2 in neural crest cells leads to aberrant enteric nervous system development in mice. *PLoS One* **13(8):**e0203391.

5. Dr. Kim's group has performed a pilot project funded through NIEHS, measuring the epigenetic stability of mammalian retrotransposon. According to the results, a small fraction of retrotransposons are hypomethylated in normal tissues, and furthermore they respond very sensitively to tumorigenesis. Using a mouse cancer model, KrasG12D, his group also measured the epigenetic instability of the imprinted genes. His group further expanded this observation by surveying the epigenetic profiles of the majority of imprinted genes in human cancers.

    a. Ekram MB, Kim J. (2014) High Throughput Targeted Repeat Elements Bisulfite Sequencing: Genome-wide DNA methylation analysis of IAP LTR retrotransposon. *PLoS ONE* **9**(7):e101683.
    b. Bakshi A, Kim J. (2014) Retrotransposon-based profiling of mammalian epigenomes: DNA methylation of IAP LTRs in Embryonic Stem, Somatic and Cancer cells. *Genomics* **104**:538-544.
    c. Bretz CL, Langohr I, Lee S, Kim J. (2015) Epigenetic instability at imprinting control regions in a KrasG12D T cell neoplasm. *Epigenetics* **10**:1111-1120.
    d. Kim J, Bretz CL, Lee S. (2015) Epigenetic instability of imprinted genes in human cancers. *Nucleic Acids Res* **43**:10689-10699.
    e. Bakshi A, Herke SW, Batzer MA, Kim J**.** (2016) DNA methylation variation of human-specific Alu elements. *Epigenetics* **11**:163-173.

Complete List of Published Work in MyBibliography:

http://www.ncbi.nlm.nih.gov/sites/myncbi/1Ls4r5kI8qnAR/bibliography/46109543/public/?sort=date&direction=ascending

D. Research Support

Ongoing Research Support

None

Pending

R21 HD118397 Kim (PI) 12/01/2025-11/30/2027
Fetal influence on maternal pregnancy through the *Peg3* imprinted domain
The goal of this study is to characterize potential gene dosage effects of the *Peg3* imprinted domain on mammalian reproduction.
Role: PI

Completed Research Support

R15 ES019118 Kim (PI) 06/01/2010-05/31/2013
Retrotransposon as a major source to epigenetic variations in the human genome
The goal of this study was to measure DNA methylation level variation of retrotransposons among individuals.
Role: PI

R01 GM066225 Kim (PI) 02/01/2004-08/31/2017
Imprinting studies of paternally expressed gene *Usp29*
The goal of this study is to characterize unknown mechanisms that are responsible for the imprinting and transcription of the *Peg3* domain.
Role: PI

R01 GM097074 Kim (PI) 06/01/2013-02/28/2019
Aebp2 as an epigenetic regulator for neural crest cells
The goal of this study is to understand Aebp2's roles in the migration and development of neural crest cells during mammalian development.
Role: PI