Min Han, Curriculum Vitae

Current Appointment:
Distinguished Professor, University of Colorado Boulder
mhan@colorado.edu.

Education:

08/1978 – 07/1982	B.S.	Biochemistry, Beijing University, Beijing
09/1983 – 09/1988	Ph.D.	Molecular Biology Institute, UCLA (with Dr. M. Grunstein)
09/1988 – 09/1991		Postdoctoral fellow, Caltech (with Dr. P. Sternberg)

Research and Professional Appointments

04/1984 – 09/1988	Thesis with Michael Grunstein at MBI, UCLA. On *Histone functions in yeast*.
09/1988 – 09/1991	Postdoctoral Fellow with Dr. Paul Sternberg, HHMI, Biology, Caltech
10/1991 – 06/1998	Assistant Professor, Dept. of MCDB, University of Colorado Boulder
07/1998 – 05/2002	Associate Professor, Dept. of MCDB, University of Colorado Boulder
07/2002 –current	Professor, Dept. of MCDB, University of Colorado Boulder
10/2000 – 06/2017	Adjunct Professor, Fudan University (Under an HHMI-CU-Fudan Agreement)
09/1997 – 10/2018	Investigator, Howard Hughes Medical Institute
1997 – current	Cancer Center Member of Health Science Center of University of Colorado
9/2019- current	Distinguished Professor, University of Colorado.

Honors and Fellowships:

1979, 1980	Outstanding Student, special Honor Outstanding Student, Beijing University
1982	Selected into CUSBEA student program to study in US
1988 – 91	Fellow of the Life Science Research Foundation
1991 – 1997	Lucille P. Markey Scholar in Biomedical Science
1992 – 1995	Basil O'Connor Scholar of March of Dimes Foundation
1993 – 1996	Searle Scholar
1997, 02, 07, 12	Selected and then renewed as a HHMI Investigator
2011	Elected to be Fellow of American Association of the Advancement of Science.
9/2019	Elected to be Distinguished Professor at University of Colorado
4/2024	Elected to be Member of American Academy of Arts and Sciences

Other External Grant Support (excluding scholarships listed above):

1996 – 2000	American Cancer Society Research Grants
1995 – 1997	March of Dimes Research Grant
1992 – current	Non-HHMI Fellowships awarded to many postdoctoral fellows in the lab
1997 – 2018	HHMI Investigator funds
11/2018	HHMI Gift Fund
1992 – 1/31/21	NIH R01GM047869
7/2019-6/2024	NIH R01AR074503
1/2020-6/2022	Colorado OEDIT AIPOC grant
1/1/21-12/31/25	NIHR35GM139631

Committee/Board Memberships (outside of CU):

1999-current	Regular and ad hoc member of multiple NIH Study Sections
various	Ad hoc member of ACS grant review committees
1999	Kavli Frontiers of Science Fellow of National Academy of Science

2006 – 2012	Associate Editor, Developmental Dynamics
2015 – 2017	Editorial Board, Worms, Genesis.
2000 – 2005	Member of Board, Director, Vice President, CBI Society
2011 – 2012	Nomination Committee, GSA
2010 – 2018	Ad hoc member of evaluation committees, Chinese Academy of Science
2011 – 2016, 2018	Biology and Medicine Panel of the Research Grants Council of Hong Kong
2018 –	Member of Scientific Advisory Board of Shanghai Institute of Nutrition and Health, Chinese Academy of Sciences
2020-2021	Member of NIH study section for R35 grant applications
Various	Member of ad hoc panels for on-site reviewing research programs at various academic institutions in US, China and Hong Kong

Major Contributions to Science as a PI:

Both as a graduate student and postdoctoral fellow, I made landmark discoveries in two different bio-frontiers of the time: epigenetics and developmental genetics. In each case, my own creative thinking was inspired by the exploratory mindset of my advisors and the intellectual freedom they granted me. Since starting my own lab in 1991, I have followed a philosophy that we should use model organisms to address outstanding and underexplored biological problems to identify new paradigms in the relevant fields. Indeed, my lab has made numerous risky shifts in research focus between distinct research fields. We have also been bold in using approaches across different disciplines and multiple model systems.

My students and postdoctoral fellows are highly encouraged to follow their own independent thinking and interests to maximize their potential to carry out innovative research. This has proven exceedingly effective in training future scientists. Despite running a modest-sized lab in Colorado, 27 of my former trainees have gone on to become professors/PIs at academic institutions. I have discussed this practice in a review article (Han 2015; Sci China Life Sci).

1. Discovery and analysis of the roles of >12 regulators of the highly-conserved RTK-RAS-MPK signaling pathway. When I established my lab in 1991, developmental genetics in worms and flies demonstrated the tremendous power in revealing the functions and mechanistic detail of major signal transduction pathways. My lab has employed several genetic suppressor screens to identify >12 factors downstream of Ras in the conserved RTK/Ras pathway, which controls developmental fate specification and cell proliferation in multicellular organisms. Our work, represented by a long list of papers, has made important contributions to the relevant fields. Several mammalian genes in the pathway, such as KSR, SUR-8 and SUR-2/MED23, bear the names from our studies in *C. elegans*. We also pioneered the chemical/genetic analysis in *C. elegans* by testing the effects of two *ras* inhibitors in 1995.

2. Established the concept of universal pairing of SUN-KASH proteins (LINC complexes) at the nuclear envelope and pioneered the study of their functions in multiple cellular/developmental events in both *C. elegans* and mice. Through genetic and molecular analysis of three genes involved in nuclear migration and anchorage, we made breakthrough findings regarding nuclear envelope proteins that mediate nucleus-related cellular functions. The Malone et al. 1999 paper (*Dev*) defined the SUN gene family after cloning the *unc-84* gene and identifying mammalian SUN1/2 proteins. The 2001 and 2002 papers (Starr et al. *Dev*; Starr and Han *Science*) defined the KASH domain and proposed the concept of the "universal" KASH-SUN pairing at the NE (LINC complexes). These published findings ignited a wave of studies on these proteins that have now become a popular research area. We also pioneered mouse genetic analysis to understand the physiological roles of SUN-

KASH complexes in muscle development, neuronal migration, gametogenesis and DNA damage responses (9 high impact papers). Five researchers who worked on these proteins have become professors/PIs.

3. Discovered the essential role of GW182 family proteins in miRISCs and developed biochemical and genetic methods to systematically analyze the *in vivo* miRNA-target interactions for specific physiological functions. Ding et al. 2005 (*Mol Cell*) was the first paper to show that a GW182 family protein is required for miRNA functions, binds to AGO and miRNAs, and brings miRISCs to P-bodies. We then pioneered the CLIP biochemical approach to systematically identify and analyze the miRNA-target interaction network under true physiological conditions, including at different developmental stages and in specific tissues (Zhang et al, 2007 *Mol Cell*; 2009 Dev; Kudlow et al., 2012, *Mol Cell*; Than et al., 2013, *Plos Genetics*). Adding combinatorial genetic tools, we have effectively uncovered important roles of many non-essential miRNAs in stress response and development.

4. Discovered unknown roles of tumor suppressors and apoptotic caspases masked by "genetic redundancy." Genetic redundancy associated with structurally unrelated genes is a common phenomenon and an impediment to the functional dissection of a genome. Over the years, my lab has tackled this problem by doing combinational genetics. The most innovative studies used two systematic approaches to identify many "hidden" functions and "redundant" genes associated with Rb and Pten (Fay et al. 2002 *Genes Dev*; Suzuki and Han 2006, *Genes Dev*). Cui et al. 2006 (*Dev Cell*) also made a breakthrough finding by showing that the SynMuvA and SynMuvB genes (including Rb) redundantly repress transcription of *lin-3*/EGF in the epidermis to prevent inappropriate cell signaling, indicating de-repression of growth factors as an important role of tumor suppressors. We later uncovered the role of Rb in regulating starvation-induced stress responses.

More recently, a "synthetic phenotype" screen led to the seminal finding of non-apoptotic and non-canonical functions of an apoptotic caspase in regulating gene expression during development (Weaver et al., 2013, *eLife*). We then uncovered an underlying mechanism and role in stress responses (Weaver et al., 2017, *Dev Cell;* Weaver et al. 2020 *Dev Cell*). Weaver (UTSW) is now a leader in the new field.

5. Uncovered multiple novel mechanisms by which animals sense the level of specific fatty acids and nucleotides to regulate animal development and behaviors. In the early 2000s, propelled by our prior analysis of human macular degeneration, which revealed a role for a fatty acid (FA) elongase, we made a bold move into the wide-open field of lipid functional analysis. In the early years, we focused our efforts on understating how specific FA variants critically influence specific cell signaling events and cellular functions. FAs are structurally diverse (>100 variants), and their levels are strictly maintained. Yet, little is known about the functional consequences of these variations, nor how animals achieve proper lipid composition in their membranes during development. The 2004 Kniazeva et al. paper (*PLoS Biol*) describes the striking, essential functions associated with the obscure but conserved monomethyl branched-chain fatty acids (mmBCFAs) (Faculty of 1000 Exceptional). Our 2012 paper described a highly innovative study showing how animals use FA variants to alter phospholipid composition at a specific stage (early embryo), which in turn specifically affects a signaling event (IP_3 signaling) for membrane dynamics. We combined complex genetics with lipid mass spectrometry and biochemistry in this extremely satisfying study (Kniazeva et al. 2012).

We then aimed to uncover mechanisms that sense the level of FA and nucleotide variants to regulate development, reproductivity, and behaviors, and discovered four such novel systems, reported in a series of high impact papers in the past few years: a TORC1-mediated intestinal system to sense the level of mmBCFA and GlcCer (through apical polarity) (Zhu et al. 2013 *eLife*; Kniazeva et al.

2015 *Dev Cell*; Zhe et al. 2015 *Genes Dev; Sewell et al. 2022 iScience*); a Notch Receptor pathway that senses the level of pyrimidine to regulate germ stem cell proliferation (Chi et al. 2016 *Genes Dev;* Jia et al. 2020 *Cell Rep*); an intestinal ATP-sensing pathway that perceives the change of vitamin B2 level to regulate protease expression and food behaviors (Qi et al. 2017 *eLife*); and acyl-CoA synthase 4 (ACS-4)–regulated myristoylation that senses the level of myristic acid to regulate sex-determination activity and the onset of oogenesis (Tang and Han 2017 *Cell*). In particular, the 2017 *Cell* paper presented a mechanism to explain a longstanding theory that fat level dictates the reproductive decision in females (reproductive adaptation). The finding of how nutrients (FA) act as environmental factors to regulate sex determination also has significant implications on the non-karyotype influence of sex determination. Moreover, the role of ACS-dependent myristoylation led us to tackle another longstanding problem: how fat deprivation leads to muscle loss (Tang et al. 2021 *Cell Rep*).

6. Made paradigm-shifting discoveries of unexpected beneficial roles of two microbial molecules on the physiology of host animals. Employing innovative genetic screens, we identified expected beneficial roles of two microbe metabolites on host physiology. First, we discovered a surprising role of the siderophore enterobactin (Ent) in promoting iron uptake and development in host animals (Qi and Han, *Cell* 2018; Sewell et al. 2024 *under review*). Mechanistically, we showed that Ent-mediated iron uptake into the host mitochondria is facilitated by Ent interaction with the ATP synthase α-subunit, which points to a novel mechanism for iron transport into mitochondria. Further studies in mammalian cells and mice suggest that such a mechanism is conserved in mammals and Ent may potentially be used as a new treatment for iron deficiency anemia. Second, we also uncovered a prominent role of bacterial cell wall derivatives, peptidoglycan (PG) muropeptides, in promoting mitochondrial homeostasis and animal development (Tian and Han, 2022 *Dev Cell*). PG muropeptides execute this role at least in part by binding and promoting the activity of ATP synthase, which points to the likely first agonist of ATP synthase. Such a role is also conserved in mammals (Tian et al.2024 *Cell Rep*).

URL to list of publications:
https://www.colorado.edu/lab/han/publications
https://www.ncbi.nlm.nih.gov/myncbi/min.han.1/bibliography/public/

Publications:

As a graduate student:

Yoshinaga, S., Dean, N., **Han, M**. and Berk, A. J. (1986). Adenovirus stimulation of transcription by RNA polymerase III: evidence for an E1A-dependent increase in transcription factor IIC concentration. **EMBO J**. 5, 343-353.

Schuster, T., **Han, M**. and Grunstein M. (1986) Yeast histone H2A and H2B amino termini have interchangeable functions. **Cell** 45: 445-451.

Han, M. Chang, M. Kim, U. and Grunstein M. (1987). Histone H2B repression causes cell cycle specific arrest in yeast: effects on chromosomal segregation, replication and transcription. **Cell** 48, 589-597.

Han, M., Kim, U., Kayne, P. and Grunstein, M. (1988). Depletion of histone H4 and nucleosomes activates the *PHO5* gene in *S. cerevisiae*. **EMBO J.**, 7, 2221-2228. PMCID: PMC454566.

Kim, U., **Han, M**., Kayne, P. and Grunstein, M. (1988). Effects of H4 depletion on the cell cycle and transcription of *Saccharomyces cerevisiae*. **EMBO, J.** 7, 2211-2219. PMCID: PMC454562.

Kayne, P., Kim, U., **Han, M**. and Grunstein, M. (1988). Extremely conserved histone H4 N-terminus in dispensable for growth but essential for repressing silent mating type genes in yeast. **Cell** 55, 27-39.

Han, M. and Grunstein, M. (1988). Nucleosome loss activates yeast downstream promoters in *vivo* in the absence of UAS elements. **Cell** 55, 1137-1145. (Cited as a key paper in 2018 Lasker Award to Michael Grunstein; Identified as one of the Gene Expression Milestone Papers by a scientist panel assembled by Nature in 2005; nature.com/milestones/geneexpressions).

Grunstein, M., **Han, M**., Kim, U., Schuster, T. and Kayne, P. (1989). Histone and nucleosome function in yeast. In *Molecular Biology of Chromosome Function*. Ed. K.W. Adolph. Springer-Verlag, New York.

As a postdoctoral fellow:

Sternberg , P. W., Hill, R., Jongeward, G., Aroian, R., **Han, M**., Mendel, J., and Holboke, A. (1989). Pattern formation during *C. elegans* vulval induction. ICN-UCLA Symp. on Dev. Biol. (Davidson et al. eds).

Han, M., Aroian, R. and Sternberg, P. (1990). The *let-60* locus controls the switch between vulval and nonvulval cell types in *C. elegans*. **Genetics**, 126, 899-913.

Han, M. and Sternberg, P. W. (1990). *let-60*, a gene that specifies cell fates during *C. elegans* vulval induction, encodes a *ras* protein. **Cell**, 63, 921-931
The two papers above, along with a paper by the R. Horvitz lab, were commented by Greenwald and Broach (Cell minireview, 1990) and Bourne et al. (1990 Nature N&V), as well as by AP news and other media outlets.

Han, M. and Sternberg, P. W. (1991). Analysis of dominant negative mutations of *C. elegans let-60 ras* gene. **Genes. & Dev.** 5, 2188-2198.

Han, M. and Sternberg, P. (1992). Pattern formation in *C. elegans*. In *Advances in Developmental Biology*. Vol. 1, 107-161. (Ed. by P. Wassarman). JAI Press.

As a Principal Investigator:

Han, M. (1992). Ras proteins in developmental pattern formation in *C. elegans* and *Drosophila.* In ***Seminars in Cancer Biol***. Vol. 3, 219-228 (ed. by D. Lowy). Acad. Press.

Han, M., Golden, A., Han, Y. and Sternberg, P (1993). The *C. elegans lin-45 raf* gene participates in *let-60 ras*-mediated vulval differentiation. **Nature**, 363, 133-140.

Han, M. (1993). Ras-mediated signaling pathway during vulval development in *C. elegans*. **Ciba Foundation symposium** 176. The GTPase superfamily. pp. 215-217.

Sternberg, P., Golden A. and **Han, M**. (1993). Role of a *raf* proto-oncogene during *C. elegans* vulval development. **Phil. Trans. Roy. Soc**., B 340, 259-265.

Sternberg, P. and **M. Han**. (1994) Let-60 ras. *Guidebook to the small GTPases*. (Eds., L.A. Huber, M. Zerial and J. Tooze).

Han, M. (1994). Common themes in different lives. Book Review on Signal Transduction- Prokaryotic and Simple Eukaryotic Systems. **Bioassay**s 16, 445-446.

Wu, Y. and **Han, M**. (1994) Suppressers of activated Ras protein defines a roles of *C. elegans* Sur-1 MAP kinase in vulval differentiation. **Genes & Dev.,** 8, 147-159.

Wu, Y., **Han, M**. & Guan, K (1995). MEK-2, a *Caenorhabditis elegans* MAP kinase kinase, functions in

Ras-mediated vulval induction and other developmental events. **Genes & Dev.**, 9, 742-755.

Hara, M. and **Han, M**. (1995). Ras-farnesyultransferase inhibitors suppress the phenotype resulting from an activated ras mutation in *C. elegans*. **Proc. Natl. Acad. Sci. USA**, 92, 3333-3337. PMCID: PMC42160

Singh, N. and **Han, M**. (1995). *sur-2*, a novel gene, functions late in the *let-60 ras*-mediated signaling pathway during *Caenorhabditis elegans* vulval induction. **Genes. & Dev.** 9, 2251-2265.

Sundaram, M and **Han, M**. (1995) The *C. elegans ksr-1* gene encodes a novel Raf-related kinase involved in Ras-mediated signal transduction. **Cell 83,** 889-901
-- Cell mini-review by J. Downward: KSR: a novel player in the RAS pathway. 15:831-4.

Sundaram, M. Yochem, J. and **Han, M.** (1996) A Ras-mediated signal transduction pathway is involved in the control of sex-myoblast migration in *Caenorhabditis elegans*. **Development.** 122, 2823-2833.

Sundaram, M. and **Han, M**. (1996). Control and integration of cell signaling pathways during *C. elegans* vulval development. **Bioassays**. 18, 473-480.

Han, M. and Sundaram, M. (1996). Ras-mediated signal transduction pathway in C. elegans. In *Regulation of the Ras signaling network*. Edited by H. Maruta and A. Burgess. R. G. Landes Company. Austin.

Zhang, K., Yeon, H., **Han, M**. & Donoso, L. A. (1996) Molecular genetics of inherited macular dystrophies. **British J. of Ophthalmology**, 80, 1018-1022.

Yochem, J. Sundaram, M. and **Han., M**. (1997). Ras is required for limited number of cell fates and not for general proliferation in Caenorhabditis elegans. **Mol. Cell. Biol.** 17, 2716-2722.

Han, M. (1997). Gut reaction to Wnt signaling in worms. **Cell** 90, 581-584.

Sugimoto, T., Stewart, S., **Han, M**. and Guan, K-L (1998). The kinase suppressor of Ras (KSR) modulates growth factor and Ras signaling by uncouples Elk-1 phosphorylation from MAP kinase activation. **EMBO J**, 17, 1717-1727. PMCID: PMC1170519

Dent, J. and **Han, M**. (1998). Post-embryonic expression pattern of *C. elegans let-60 ras* reporter constructs. **Mech. of Devel.** 72, 179-182.

Yochem, J., Gu, T. and **Han, M**. (1998) A new marker for mosaic analysis of *C. elegans* suggests an interconnection between hyp6 and hyp7, two major components of the epidermis**. Genetics**, 149:1323-34.

Gu, T., Orita S., and **Han, M.** (1998) *C. elegans* SUR-5, a novel but conserved protein, negatively regulates LET-60 Ras activity during vulval induction. **Mol. Cell. Biol**. 18:4556-4564.

Sieburth, D., Sun, Q. and **Han, M**. (1998). SUR-8, a conserved Ras-binding protein with leucine-rich repeats, positively regulate Ras-mediated signaling in *C. elegans*. **Cell**. 94, 119-130
-- Cell review by Sternberg and Alberola: Conspiracy theory: RAS and RAF do not act alone. 13:447-50

Sternberg, P. and **Han, M**. (1998). Genetics of Ras Signaling in *Caenorhabditis elegans*. **Trends in Genetics**.14, 466-472.

Hanna-Rose, W. and **Han, M.** (1999). Cog-2, a Sox domain protein necessary for establishing the functional connection between the uterus and the vulva in *Caenorhabditis elegans*. **Development**. 126, 169-179.

Yochem, J. Tucker, S., Greenwald, I. and **Han, M**. (1999). A gp330/megalin-related protein is required in the major epidermis of *Caenorhabditis elegans* for completion of molting. **Development** 126, 597-606.

Malone, C. J., Fixen, W. D., Horvitz, H. R. and **Han, M**. (1999) UNC-84 localizes to the nuclear envelope and is required for nuclear migration and anchoring during *C. elegans* development. **Development**. 126, 3171-3181. (First defined SUN family proteins and identified mammalian SUN1/2)

Guan, K. and **Han, M**. (1999). A G-protein signaling network mediated by RGS. **Genes & Dev.** 13, 1763-1767.

Stewart, S., Sundaram, M., Zhang, Y., Lee, J., Xiong, Y. **Han, M**., and Guan, K-L. (1999). Kinase Suppressor of Ras (KSR) forms a multi-protein signaling complex and modulates MEK localization. **Mol. Cell. Biol.** 19, 5523-5534. PMCID: PMC84397.

Sieburth, D. Sundaram, M. Howard, R. M. and **Han, M.** (1999). A PP2A regulatory subunit positively regulates Ras-mediated signaling during C. elegans vulval induction. **Genes & Dev**. 13, 2562-2569. PMCID: PMC317062.

*Kniazeva, M., Chiang, M. F., Cutting, G. R., Zack DJ., **Han, M**., Zhang, K. (1999). Clinical and genetic studies of an autosomal dominant cone-rod dystrophy with features of Stargardt disease. **Ophthalmic Genetics** 20, 71-81.

*Kniazeva, M., Chiang, M. F., Morgan B, Anduze AL, Zack DJ, **Han, M**., Zhang, K. (1999) A new locus for autosomal dominant Stargardt-like disease maps to chromosome 4. **Amer. J. Hum. Gen**. 64, 1394-1399. PMCID: PMC1377876.

*Zhang K, Garibaldi DC, Kniazeva M, Albini T, Chiang MF, Kerrigan M, Sunness JS, **Han M**, Allikmets R. (1999). A novel mutation in the ABCR gene in four patients with autosomal recessive Stargardt disease. **Am J Ophthalmol.** 128:720-4

*Zhang K, Kniazeva M, Hutchinson A, **Han M**., Dean M, Allikmets R. (1999) The ABCR Gene in Recessive and Dominant Stargardt Disease: A genetic Pathway in Macular Degeneration. **Genomics,** 60:234-237.

*Kniazeva M, Traboulsi EI, Yu Z, Stefko ST, Gorin MB, Shugart YY, O'Connell JR, Blaschak CJ, Cutting G, **Han M,** Zhang K. (2000). A new locus for dominant drusen and macular degeneration maps to chromosome 6q14. **Am J Ophthalmol**. 130:197-202.

*Zhang K, Kniazeva M, **Han M**, Li W, Yu Z, Yang Z, Li Y, Metzker ML, Allikmets R, Zack DJ, Kakuk, LE, Lagali PS, Wong PW, MacDonald IM, Sieving PA, Figueroa DJ, Austin CP, Gould RJ, Ayyagari R, Petrukhin K. (2001). A 5-bp deletion in ELOVL4 is associated with two related forms of autosomal dominant macular dystrophy. **Nature Genetics**, 27, 89-93.

* *Kniazeva: a member of the laboratory.*

Fay, D. S., Stanley, H., **Han, M**. and Wood, W. B. (1999) A *C. elegans hunchback* homologue is required for late but not early embryonic patterning. **Devel. Biol.** 205, 240-253.

Fay, D.S. and **Han, M**. (2000). The Synthetic Multivulval Genes of *C. elegans*: Functional redundancy, Ras-antagonism, and cell fate determination. **Genesis**. 26, 279-284.

Fay, D. S. and **Han, M**. (2000). Mutations in cye-1, a *C. elegans* cyclin E gene homolog, reveal coordination between cell-cycle and vulval development. **Development**,127, 4049-4060.

Li, W., **Han, M**. and Guan, K.L. (2000). The Leucine-rich repeat protein SUR-8 enhances MAP kinase activation and forms a complex with Ras and Raf. **Genes & Dev**.14, 895-900. PMCID: PMC316541

Grant, K, Hanna-Rose, W. and **Han, M**. (2000). *Sem-4* promotes *Caenorhabditis elegans* vulval fate determination through regulation of *lin-39* hox. **Devel. Biol**. 224, 496-506.

Han, M. (2000). Studies on Ras-mediated signal transduction in *C. elegans*. In *Stem Cells and Development*. Ed. By J. Ye, T. Xu, Tang X, and Bei H. Medical Science Press. Beijing, China.

Chen, Z. and **Han, M**. (2000). Building a protein interaction map: research in the post-genomic era. **BioEssays**, 22, 503-506.

Hanna-Rose, W. and **Han, M**. (2000). Getting signals crossed in *C. elegans*. **Curr. Opin. Devel. Genet**. 10, 523-528.

Yoder, J. and **Han, M**. (2001). Cytoplasmic dynein light intermediate is required for discrete aspects of mitosis in *Caenorhabditis elegans*. **Mol. Cell. Biol.** 12, 2921-2933. PMCID: PMC60145.

Chen, Z. and **Han, M.** (2001). Role of Rb/E2F, the NuRD Complex, and Ras in Regulating a *lin-39* Hox-mediated Cell Fusion Process during Vulval Fate Specification in *C. elegans* **Curr. Biol.** 11, 1874-1879.

Chen, Z., **Han**, M. (2001) Role of *C. elegans lin-40* MTA in vulval fate specification and morphogenesis. **Development**. 128, 4911-4921.

Spencer, A., Orita, S., Malone, C., and **Han, M**. (2001) A RHO GTPase –mediated pathway is required during P cell migration in *C. elegans*. **Proc. Natl. Acad. Sci. USA**. 98, 13132-13137. PMCID: PMC60836.

Starr, D, Hermann, GJ., Malone, C. Fixsen, W., Priess, J. Horvitz, HR and **Han, M.** (2001). *unc-83* encodes a novel component of the nuclear envelope and is essential for proper nuclear migration. **Development.** 128, 5039-5050.

Lee, K.K*, Starr, D*. Cohen, M., Liu, J., **Han, M**. Wilson, K., and Gruenbaum, Y. (2002) UNC-84 requires Ce-lamin for its nuclear envelope localization. **Mol. Cell Biol**, 13, 892-901. *co-first authors, Starr was a postdoctoral fellow in the lab. PMCID: PMC99607.

Hanna-Rose, W. and **Han, M**. (2002). The *Caenorhabditis elegans* EGL-26 protein mediates vulval cell morphogenesis. **Devel. Biol**. 24, 247-258. (Cover photo)

Antoshechkin, I. and **Han, M**. (2002). *Caenorhabditis elegans evl-20* gene encodes a functional homologue of human small GTPase ARL2 and regulates cytoskeleton dynamics during cytokinesis and morphogenesis. **Developmental Cell**, 2, 579-591.

Suzuki, Y., Morris, G., **Han**, M. and Wood, W.B. (2002). A cuticle collagen encoded by the lon-3 gene may be a target of TGF-beta signaling in determining *Caenorhabditis elegans* body shape. **Genetics**, 162 1631-1639.

Starr, D. and **Han, M**. (2002) Role of ANC-1 in Tethering Nuclei to the Actin Cytoskeleton. **Science.** 298, 406-409. Faculty of 1000 Must Read.

Fay, D., Keneen, S. and **Han, M**. (2002) *fzr-1* and *lin-35*/Rb Function Redundantly to Control Cell Proliferation in *C. elegans* as Revealed by a Nonbiased Synthetic Screen. **Genes & Dev**. 16. 503-517. (Cover illustration). Faculty of 1000 Must Read.

Fay, D., Large, E. **Han, M**. and Darland, M. (2003) *lin-35*/Rb and *ubc-18*, an E2 ubiquitin-conjugating enzyme, function redundantly to control pharyngeal morphogenesis in *C. elegans*. **Development.** *130, 3319-30.*

Kniazeva, M., Sieber, M. McCauley. S. Zhang, K. Watts, J., **Han, M**. (2003). Suppression of *C. elegans* ELO-2 function results in disruption of palmitic acid elongation and causes multiple physiological defects including abnormal ultradian rhythms, in *Caenorhabditis elegans*. **Genetics**, 163 159-169.

Starr, D and **Han, M**. (2003) ANChors away: an actin based mechanism of nuclear positioning. **Journal of Cell Science**. 15;116:211-6.

Cui, M. and **Han. M**. (2003) *Cis* Regulatory Requirements for Vulval Cell-Specific Expression of the *Caenorhabditis elegans* Fibroblast Growth Factor Gene *egl-17***. Dev. Biol**. 257, 104-116.

Wang, F., Yoder, J., Antoshechkin, I, and **Han, M**. (2003). *C. elegans* EVL-14/PDS-5 and SCC-3 are essential for sister chromatid cohesion in mitosis and meiosis. **MCB**, 23, 7698-7707. PMCID: PMC207601.

Han, M. *C. elegans* genetics (2004). A chapter in the online Genetics textbook by Virtual Text (ergito.com).

Yoder, J.H., Chong, H., Guan, K.L, and **Han, M.** (2004). Modulation of KSR activity in *C. elegans* by Zn ions, PAR-1 kinase and PP2A phosphatase, **EMBO J**. 23, 111-119. PMCID: PMC1271663.

Eastburn, D. and **Han, M**. (2004). When Ras Signaling Reaches the Mediator (2004). **Developmental Cell**. 6 158-159. PMID: 14960267

Bourbon HM and 44 other authors (2004). A unified nomenclature for protein subunits of mediator complexes linking transcriptional regulators to RNA polymerase II. ***Mol. Cell*** 14(5):553-557, 2004.

Cui, M., Fay, DS and **Han,** M. (2004). *lin-35/Rb* cooperates with the SWI/SNF complex to control *Caenorhabditis elegans* Larval Development. **Genetics**, 167, 1177-85. PMCID: PMC1470958.

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