BIOGRAPHICAL SKETCH

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NAME: David Katz

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

POSITION TITLE: Associate Professor, Dept. of Cell Biology

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
Washington University in St. Louis	B.A.	05/97	Biology
Princeton University, Princeton, NJ	Ph.D.	01/05	Molecular Biology
Emory University, Atlanta, GA	Postdoctoral	2004-10	Developmental Genetics

B. Positions, Scientific Appointments, and Honors

2018- Associate Professor, Department of Cell Biology, Emory University, Atlanta, GA
2010-2018 Assistant Professor, Department of Cell Biology, Emory University, Atlanta, GA
2004-2010 Postdoc, Dept. of Biology, Emory University, Atlanta, GA Advisor: Dr. William Kelly
2000-2004 Ph.D. Molecular Biology, Princeton University, Princeton, NJ Advisor: Dr. Shirley Tilghman
1997 B.A. in Biology, Washington University, St. Louis, MO

Other Experience and Memberships

2024- Elected as Director of the Genetics and Molecular Biology Graduate Program at Emory University
2024 National Chair of the C. elegans portion of the Genetics Society of America Allied Genetics Meeting, March 2024, Washington DC
2023- Elected to the national *C. elegans* Wormboard
2022 Co-Chair 6th International Conference on Epigenetics and Bioengineering
2021 Organizing Committee 5th International Conference on Epigenetics and Bioengineering
2021-25 Elected as basic sciences representative to the Dean's Faculty Advisory Committee, Emory University School of Medicine
2019- Society for Neuroscience
2018 Guest Editor: PLoS Genetics
2016-22 American Cancer Society: DDC Co-Chair and Study Section Member
2011-18 Reviewer: Biology of Reproduction, Molecular Reproduction and Development, Developmental Cell, PLoS Genetics, Epigenetics and Chromatin, eLife, Cell Reports, Worm, Basic and Clinical Andrology, Nature, PNAS, BMC Biology, EMBO Journal, Nature Communications, iScience, Development, Biochemical Society Transactions, Asian Journal of Andrology, NAS, Cytotechnology, Review Commons, Nature Structural and Molecular Biology, Scientific Reports, Genetics, Life Science Alliance, Wormbook, Mechanisms of Ageing and Development, Brain
2007- American Society for Cell Biology
2006- Society for Developmental Biology

Honors

2021 Invited Plenary talk, 1st ever DEI session at the International Worm Meeting on Co-director of the Pipeline CURE program at Oglethorpe University: Lowering institutional barriers to research by reiteratively incorporating original *C. elegans* experiments throughout a biology curriculum.
2021 Selected finalist for the National Postdoc Association Gallagher Mentor Award (1 of 8 nationally)
2019 Escher Prize in Germ Cell Exposures for pioneering how defects in maternal epigenetic

reprogramming can give rise to neurodevelopmental disorders.
2018 Emory Millipub Award given to researchers with a publication that has been cited 1000 times
2018 Emory School of Medicine Researcher Appreciation Day Recognition: Highlighted on Special Emory Website
2012 Outstanding Postdoctoral Fellow "one in one hundred" Mentor Award Emory University SOM
2006-09 NIH Postdoctoral F31 NRSA

Presentations (selected or invited speaker)

Seminars: (2025) UCSF Audacious Science Lecture, Princeton University Molecular Biology, University of Iceland Reykjavik School of Health Sciences, Rutgers University Worm Supergroup, Emory University Genetics Grand Rounds **(2024)** Kabuki Syndrome Foundation, Georgia State University, The University of Pittsburgh Worm Meeting, **(2023)** The University of Pennsylvania Epigenetics Institute, **(2021)** The University of Texas Southwestern, Dept. of Molecular Biology, The University of Georgia, Dept. of Physiology and Pharmacology, **(2020)** NIH NIDDK, **(2019)** University of Massachusetts Medical School, Neuroscience Program, **(2018)** Seattle Children's Hospital, Dev. Biology and Regenerative Medicine, Stony Brook University, Dept. of Neurobiology, MD Anderson Cancer Center Dept. of Molecular Carcinogenesis, **(2017)** Washington University in St. Louis, Dept. of Neuroscience, **(2016)** Emory University, Dept. of Human Genetics, The University of Georgia, Genetics Dept., **(2015)** University of Texas San Antonio, Dept. of Biology, **(2014)** Institut Curie/IBENS, Paris France, IGBMC Strasbourg France, Georgia Institute of Technology, **(2013)** Kennesaw State University, Dept. of Biology, University of Florida, Dept. of Molecular Genetics and Microbiology, **(2012)** Emory University, Winship Cancer Center, Georgia State University, Biology Dept., **(2011)** Emory University, Dept. of Biochemistry, **(2010)** Stony Brook University, Dept. of Molecular Genetics and Microbiology, Princeton University, Dept. of Molecular Biology

Meetings: (2025) Invited Distinguished Alumni Speaker Princeton University: Many Minds Many Stripes Conference, Invited speaker Epigenetics Gordon Research Conference **(2024)** Invited speaker SDB annual meeting **(2021)** Plenary talk on DEI initiative, International Worm Meeting, Alzheimer's and Parkinson's Disease Conference **(2020)** Physiological Irrelevant Conference, Tel Aviv Israel **(2019)** Keynote speaker Florida Worm Meeting **(2018)** Cold Spring Harbor Germ Cell Meeting, Neurobiology of Brain Disorders Gordon Research Conference, Cold Spring Harbor Laboratory Mouse Course, Fusion Epigenetics Conference, Cancún Mexico, **(2017)** Epigenetics Gordon Research Seminar, Discussion Leader, **(2016)** Epigenomics of Common Diseases Conference, Cambridge, UK, Society for Developmental Biology, National Meeting, Boston, The Allied Genetics Conference, Orlando, **(2015)** Epigenetics Gordon Research Conference, **(2014)** Cold Spring Harbor Germ Cell Meeting speaker/session chair, Southeast Regional Society for Developmental Biology Meeting speaker/session chair, **(2013)** Select Biosciences Epigenetics Meeting, Boston, **(2012)** *C. elegans* Development, Cell Biology and Gene Expression, **(2011)** Keystone Symposia "Epigenomics"

Current Trainees
Graduate Students: Yu Bai (2020-), Saahj Gosrani (2021-) Monica Reeves (2022-), Mackenzie Roberson (2025-), Caitlin Fix (2025-),
Postdoctoral Fellows: Dr. Miguel Soares (2025-)

Former Trainees
Graduate Students: Dr. Jadiel Wasson (2011-16) Currently tenure track Assistant Professor NYU, Dr. Michael Christopher (2012-16) Currently Staff Scientist Emory University Epigenomics Core, Dr. Amanda Engstrom (2015-20) Currently: Postdoctoral Fellow w/ Dr. Huda Zogbhi, Baylor/HHMI, Dr. Alyssa Scott (2016-22) Currently Grants Management, SUNY Buffalo, Dr. Juan Rodriguez (2017-23) Currently Scientist at Genentech
Postdoctoral Fellows: Dr. Shana Kerr (2010-12) Currently Principal Academic Professional (teaching professor) Georgia Tech, Dr. Ernest Ricks (2011-14) Currently Director of Graduate Education Morehouse School of Medicine, Dr. Teresa Lee (2014-20) Currently: Tenure track Assistant Professor UMass-Lowell, Dr. Brandon Carpenter (2015-21) Currently Tenure track Assistant Professor Kennesaw State University, Dr. Onur Birol (2017-21) Currently Academic Professional (teaching professor) Georgia Tech

C. Contributions to Science

1. Following the elucidation of cis-regulatory elements in the Hox cluster of *Drosophila*, it was subsequently shown that the ability to control segmentation is heritably controlled during development by trans regulatory factors, such as TRITHORAX and POLYCOMB. Many years later the discovery of histone modifications and the identification of the original trans regulatory factors as histone modifying enzymes suggested the possibility that histone modifications could play a mitotically heritable role in controlling gene

regulation. Nevertheless, despite the efforts of countless labs, good evidence for this role remained elusive. In experiments with a *C. elegans Lsd1*/*Kdm1*a ortholog, *spr-5*, we demonstrated that the loss of function of this gene results in a germline mortality phenotype in which the incidence of sterility progressively increases across generations, due to the transgenerational accumulation of the histone modification H3K4me2 (paper A). This provided some of the best evidence to date that histone methylation can be mitotically heritable. Moreover, this data extended this paradigm by suggesting that histone methylation can potentially be transmitted across generations and is normally reprogrammed between generations via the action of histone modifying enzymes. In follow up work, we showed that worms lacking the H3K4 methyltransferase component WDR-5, or the H3K9 demethylase JHDM-1, progressively live longer over 20 generations due to the accumulation of the repressive histone modification H3K9me2 (paper B). This second example of transgenerational inheritance mediated by histone methylation reinforces the paradigm and demonstrates that complex traits can be modulated by the accumulation of both active and repressive histone methylation.

a. **Katz D.J.,** Edwards M.T., Reinke V. and Kelly W.G. A. *C. elegans* LSD1 demethylase contributes to germline immortality by reprogramming epigenetic memory. 2009. *Cell,* **137**: 308-320. PMID: 19379696.

-Comment in: *Cell,* **137**: 203-205.

-Recommended by Faculty of 1000

b. Lee T.W., David H.S., Engstrom A.K, Carpenter B.S. and **Katz D.J.** Repressive H3K9me2 protects lifespan against the transgenerational burden of COMPASS activity in *C. elegans*. 2019. *eLife.* Dec. 9;8:e48498 PMID: 31815663.

-Comment in *eLife* 2020;9:e54296

-Recommended by Faculty of 1000

2. Since the discovery of the first histone modifications by the Allis Lab, there has been intense interest in the idea of a histone code, where combinations of histone modifications act coordinately to regulate chromatin and the expression of genes contained within that chromatin. A number of labs have provided evidence for this type of coordinate regulation in cells. Nevertheless, evidence for this type of coordinate regulation of histone modifications during developmental processes remains elusive. In paper A, we demonstrate that H3K4 and H3K9 methylation are coordinately regulated in *C. elegans* by the H3K4me2 demethylase LSD1 and the H3K9 methyltransferase, MET-2. These enzymes form a reprogramming switch (from euchromatin to heterochromatin) at fertilization that prevents germline genes from the previous generation from continuing to be expressed inappropriately in the subsequent generation, and this reprogramming is essential to maintain the ability to properly specify the germline in the subsequent generation. This work provides some of the best evidence to date of how histone modifications are coordinately regulated developmentally. In addition, this work provides evidence of a broader reprogramming mechanism that exists between generations to regulate critical histone information. In paper B, we subsequently showed that the ectopic expression of germline genes in somatic cells, due to failure to reprogram histone methylation between generations, causes developmental delay by enabling the inappropriate retention of a third histone modification, H3K36me3, in somatic tissues. This work provides an elegant example of how an organism finely balances multiple histone modifications to help properly specify cell fates. In paper C, we found that the ectopic expression of germline genes in neurons causes a severe chemotaxis behavior defect. But surprisingly, the invariant *C. elegans* embryonic lineage and adult nervous system were intact in the progeny of *spr-5; met-2* double mutants and the chemotaxis defect could be rescued by shutting off the ectopic transcription of germline genes even after the nervous system was already completely formed. This suggests that ongoing inappropriate transcription can block the function of neurons. If this were to also be the case in the corresponding human patients, it might be possible to rescue cognitive defects in these patients by shutting off the inappropriate transcription in neurons. In paper D, we went on to show that loss of LSD1 maternally in mice results in arrest prior to the 2-cell stage of embryogenesis with arrested embryos failing to undergo the maternal to zygotic transition in transcription. This demonstrates that the maternal role of LSD1 in epigenetic reprogramming is conserved and is absolutely required for the progression of embryogenesis in mammals. Moreover, we find that partial loss of LSD1 maternally results in striking phenotypes weeks after fertilization, including perinatal lethality and abnormal behavior in surviving adults. This work establishes a novel mammalian paradigm where defects in early maternal reprogramming can lead to defects that manifest later in development. This potential for defects in maternal reprogramming to contribute to defects in the adult has recently been recognized by Dr. Katz receiving the Escher Fund for Autism- Prize in Germ Cell Exposures.

a. Kerr S.C.*, Ruppersburg C.C*, Francis J and **Katz D.J.** (*these authors contributed equally) SPR-5 and MET-2 function cooperatively to reestablish an epigenetic ground state during passage through the germline. *PNAS* 2014 July 1;111(26):9509-14. PMID: 24979765.
b. Carpenter B.S., Lee T.W, Plott C., Brockett J., Myrick D.A. and **Katz D.J.** *C. elegans* establishes germline versus soma by balancing inherited histone methylation. *Development*. 2021 Feb 10;148(3) PMID: 33462111
-featured in *Development's* people behind the papers
c. *Rodriguez J.D., *Reeves M.N, Chavez, S.R.,Wang H.L., Chavez, J.Z, Rastogi R, Sun, L.I., Roberson M.S., Preston E.A., Massenburg Z., Cruz, K.N., Chadha M.S., Hill M.J., Soares, M.L., Corces V.G., Schmeichel K.L., Murray J.I. and **Katz D.J** Ectopic transcription due to inappropriately inherited histone methylation may interfere with the ongoing function of terminally differentiated neurons. *PNAS.* 2025 Sep 30;122 (39) (*Authors contributed equally) PMID: 40991443
d. Wasson J.A., Simon A.K., Myrick D.A., Wolf G., Driscoll S., Pfaff S.L., MacFarlan T.S. and **Katz D.J.**. Maternally provided LSD1 enables the maternal-to-zygotic transition and prevents defects that manifest postnatally. 2016 *eLife*, Jan. 27;5:e08848. PMID: 26814574.

3. Since Alois Alzheimer first discovered pathological aggregates in the brains of demented patients, it has been known that these aggregates correlate with disease. However, over 100 years later it remains unknown how these aggregates lead to neuronal cell death. In paper A, we have uncovering a novel epigenetic step in Alzheimer's disease (AD) that links pathological aggregates to neuronal cell death. We have found that mice in which the histone demethylase *Lsd1* is inducibly deleted as adults recapitulate most aspects of AD, including widespread hippocampal and cortex neuronal cell death, learning and memory deficits, as well as transcriptional changes that match the human patients genome-wide. In addition, we have shown that LSD1 is specifically mislocalized to pathological tau aggregates in the brains of AD patients. Furthermore, in paper B we have provided direct evidence that tau functions to kill neurons through the sequestration of LSD1. This has lead to the discovery that the overexpression of LSD1 blocks tau mediated neurodegeneration in a mouse even after the formation of Tau aggregates. Based of this evidence, we have proposed the following model: as neurons age, the accumulation of protein aggregates sequesters LSD1 in the cytoplasm and interferes with the continuous requirement for LSD1 in the nucleus. Normally, LSD1 maintains terminally differentiated neurons, and prevents the activation of common neurodegenerative pathways, by continuously repressing the transcription of inappropriate genes. As a result, the inhibition of LSD1 by pathological aggregates in the aging neurons of AD brains creates a situation where neurons are subject to an onslaught of detrimental processes. This results in neuronal cell death and dementia. This previously unknown epigenetic step linking pathological aggregates to the downstream pathways in AD provide an ideal new drug target. In addition, the requirement for LSD1 in differentiated neurons fundamentally alters our view of differentiated cells by suggesting that they must continuously employ epigenetic mechanisms to homeostatically maintain their cell fate.
a. Christopher, M.A., Myrick, D.A., Barwick, B.G., Engstrom, A., Porter-Stransky, K.A., Boss, J.M., Weinshenker, D., Levey, A.I. and Katz, D.J. LSD1 continuously protects against hippocampal and cortical neurodegeneration. 2017 8(1):805 *Nature Communications*. PMID: 28993646
b. Engstrom, A., Walker, A., Moudgal, R.A., Myrick, D.A., Kyle, S.M., Bai, Y., Rowley J.M. and **Katz, D.J.** The inhibition of LSD1 via sequestration contributes to tau-mediated neurodegeneration. 2020 Nov 17;117(46):29133-29143 *PNAS*. PMID: 33139560

Current list of publications
https://www.ncbi.nlm.nih.gov/sites/myncbi/1nE1doT1gp_/bibliography/49163341/public/?sort=date&direction=ascending