BIOGRAPHICAL SKETCH

NAME: Jones, Kenneth Lloyd

eRA COMMONS USER NAME: KL.JONES

POSITION TITLE: Associate Professor

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE	Completion Date MM/YYYY	FIELD OF STUDY
Colorado State University	BS	05/1995	Wildlife Biology
Texas A&M University – Kingsville	MS	08/1998	Wildlife Biology
University of Illinois – Chicago	PhD	07/2003	Biology (Genetics)
Kansas State University	Postdoctoral	05/2007	Ecological Genomics
University of Georgia	Postdoctoral	06/2009	Environmental Genomics

A. Personal Statement

In addition to my own research, a major component of my current position at the University of Colorado is to collaborate with other researchers, where I assist in the design, implementation, and analysis of next-generation sequencing data. In that capacity, I was appointed as the Director of Bioinformatics for the Colorado Cancer Center's Biostatistics and Bioinformatics Shared Resource (2012-2017), where I applied RNAseq and Exome-seq to multiple projects spanning many areas of research, a short list include: cancer, cardiovascular disease, obesity, viral latency, autophagy, dermatological disease. For this proposal, I will continue to provide bioinformatic contributions to this project in both analysis of new data and mining of published data to put this data in context. In particular, my role will be to help with the mapping of the sequence data, provide advice on optimal analytical tools for the biological questions relevant to this project, query available databases to uncover gene expression signatures that will help develop hypotheses stemming from the proposed work. For this project, we will utilize custom bioinformatic pipelines currently running on our server. This server contains a sufficiently large amount of RAM (up to 3/4 TB of RAM on a single node) to enable the analysis proposed. On this server, we can also setup and compare new pipelines as they become available. I have recently been recruited to the University of Oklahoma School of Medicine as Associate Professor in Cell Biology, as wel as appointed the Director of the core facilities (Genomics, Proteomics, Cytometry, Confocal Imaging, and Bioinformatics). In addition to my new duties at OU, I will continue this and other valuable UC Denver collaborations from my new position at OU.

B. Positions and Honors

Positions and Employment

2003-2007 Postdoctoral Research Associate, Ecological Genomics Institute, Division of Biology, Kansas State University, Manhattan, KS.

2007-2008 Postdoctoral Research Associate, Savannah River Ecology Lab, University of Georgia, Aiken SC.

2008-2009 Postdoctoral Research Associate, Dept of Environmental Health Science, University of Georgia, Athens, GA.

2009-2011 Assistant Research Scientist, University of Georgia, Georgia Genomics Facility and the Dept of Environmental Health Science, Athens, GA.

2009 to present Adjunct Professor, Department of Biological Sciences, University of New Orleans, New Orleans, LA.

2011 to 2016 Assistant Research Professor, University of Colorado School of Medicine, Dept of Biochemistry and Molecular Genetics, Aurora, CO.
2011 to 2017 Director of Bioinformatics, Biostatistics and Bioinformatics Shared Resource, University of Colorado Cancer Center, Aurora, CO.
2016 to 2018 Assistant Research Professor, University of Colorado School of Medicine, Dept of Pediatrics, Section of Hematology, Oncology, and Bone Marrow Transplant, Aurora, CO.
2016 to 2019 Program Leader in Genomics and Bioinformatics, University of Colorado School of Medicine, Dept of Pediatrics, Section of Hematology, Oncology, and Bone Marrow Transplant, Aurora, CO.
2018 to 2019 Associate Director for Bioinformatics, Colorado Nutrition Obesity Research Center, University of Colorado School of Medicine, Aurora, CO.
2018 to 2019 Associate Director for Bioinformatics, Skin Diseases Research Center Research Center, University of Colorado School of Medicine, Aurora, CO.
2018 to 2019 Associate Professor, University of Colorado School of Medicine, Dept of Pediatrics, Section of Hematology, Oncology, and Bone Marrow Transplant, Aurora, CO.
2019 to present Associate Professor, University of Oklahoma School of Medicine, Dept of Cell Biology, Oklahoma City, OK.
2019 to present Director, Laboratory for Molecular Biology and Cytometry Research, University of Oklahoma School of Medicine, Oklahoma City, OK.
2019 to present Associate Director for Bioinformatics, Laboratory for Molecular Biology and Cytometry Research, University of Oklahoma School of Medicine, Oklahoma City, OK.
2019 to present Director for Bioinformatics, Harold Hamm Diabetes Center, University of Oklahoma School of Medicine, Oklahoma City, OK.
2019 to present Director for Bioinformatics, Stephenson Cancer Center, University of Oklahoma School of Medicine, Oklahoma City, OK.

Other Experience and Professional Memberships

1996-2015 Genetic Advisor and Genealogist to the joint US Fish and Wildlife Service and Canadian Wildlife Service Whooping Crane Recovery Team.
2004-2015 Genetic Advisor and Genealogist to the US Fish and Wildlife Service Masked Bobwhite Quail Recovery Team.
2003-2007 Member and trainee of the Ecological Genomics Institute at Kansas State Univ

C. Contribution to Science

Throughout my career, I have focused on collaborative multi-investigator research where I have applied genetic techniques to solve specific biological questions. In addition to my own research, a major component of my current position at the University of Colorado is to collaborate with other researchers, where I assist in the design, implementation, and analysis of next-generation sequencing data. Recent contributions can be arranged into applications of NGS methodologies.

1. **Cancer Genomics:** We are using NGS to determine the causal variants for, and major pathways involved in various types of cancers (solid tumors and blood cancers).
   a. McCoach CE, Le AT, Gowan K, Jones K, Schubert L, Doak A, Estrada-Bernal A, Davies KD, Merrick DT, Bunn PA Jr, Purcell WT, Dziadziuszko R, Varella-Garcia M, Aisner DL, Camidge DR, Doebele RC. Resistance Mechanisms to Targeted Therapies in *ROS1*$^{+}$ and *ALK*$^{+}$ Non-small Cell Lung Cancer. Clin Cancer Res. 2018 Jul 15;24(14):3334-3347. doi: 10.1158/1078-0432.CCR-17-2452. Epub 2018 Apr 10. PubMed PMID: 29636358; PubMed Central PMCID: **PMC6050099**.
   b. Lummus SC, Donson AM, Gowan K, Jones KL, Vibhakar R, Foreman NK, Kleinschmidt-DeMasters BK (2017) p16 Loss and E2F/cell cycle deregulation in infant posterior fossa ependymoma. Pediatr Blood Cancer. 64(12). PubMed PMID: 28548702; PubMed Central PMCID: **PMC5647247**.
   c. Fernández-Cabezudo MJ, Faour I, Jones K, Champagne DP, Jaloudi MA, Mohamed YA, Bashir G, Almarzooqi S, Albawardi A, Hashim MJ, Roberts TS, El-Salhat H, El-Taji H, Kassis A, O'Sullivan DE,

Christensen BC, DeGregori J, al-Ramadi BK, Rincon M (2016) Deficiency of mitochondrial modulator MCJ/DNAJC15 promotes chemoresistance in breast cancer. *JCI Insight* May 19;1(7) **PMC4888911**

d. Noetzli L, Lee-Sherick AB, Callaghan M, Noris P, Rajpurkar M, Jones K, Gowan K, Savoia A, Balduini C, Pecci A, Gnan C, De Rocco D, Doubek M, Li L, Lu L, Leung R, Lo R, Landolt-Marticorena C, Hunger S, Heller P, Gutierrez Hartman A, Xiayuan L, Pluthero FG, Rowley J, Weyrich AS, Kahr WHA, Porter CC, Di Paola J. (2015). Germline mutations in ETV6 are associated with thrombocytopenia, red cell macrocytosis and predisposition to lymphoblastic leukemia. *Nature Genetics*, May;47(5) 535-8. **PMC4631613**

2. **DNASeq:** We are using DNASeq (whole genome, exome, and targeted sequencing) in humans to determine the causal variants for major medical phenotypes/diseases. We have applied DNASeq to several disorders including: cancer, scoliosis, blood disorders, mitochondrial disorders, heart disease, and others.
   a. Begay RL, Graw SL, Sinagra G, Asimaki A, Rowland TJ, Slavov DB, Gowan K, Jones KL, Brun F, Merlo M, Miani D, Sweet M, Devaraj K, Wartchow EP, Gigli M, Puggia I, Salcedo EE, Garrity DM, Ambardekar AV, Buttrick P, Reece TB, Bristow MR, Saffitz JE, Mestroni L, Taylor MRG. Filamin C Truncation Mutations Are Associated With Arrhythmogenic Dilated Cardiomyopathy and Changes in the Cell-Cell Adhesion Structures. JACC Clin Electrophysiol. 2018 Apr;4(4):504-514. doi: 10.1016/j.jacep.2017.12.003. Epub 2018 Feb 2. PubMed PMID: 30067491; PubMed Central PMCID: **PMC6074050**.
   b. Astling DP, Heft IE, Jones KL, Sikela JM. High resolution measurement of DUF1220 domain copy number from whole genome sequence data. BMC Genomics. 2017 Aug 14;18(1):614. doi: 10.1186/s12864-017-3976-z. PubMed PMID: 28807002; PubMed Central PMCID: **PMC5556342**.
   c. Davies KD, Farooqi MS, Gruidl M, Hill CE, Woolworth-Hirschhorn J, Jones H, **Jones KL**, Magliocco A, Mitui M, O'Neill PH, O'Rourke R, Patel NM, Qin D, Ramos E, Rossi MR, Schneider TM, Smith GH, Zhang L, Park JY, Aisner DL. Multi-Institutional FASTQ File Exchange as a Means of Proficiency Testing for Next-Generation Sequencing Bioinformatics and Variant Interpretation. J Mol Diagn. 2016 Jul;18(4):572-9. doi: 10.1016/j.jmoldx.2016.03.002. Epub 2016 May 4. PubMed **PMID: 27155050**.
   d. Baschal EE, Wethey CI, Swindle K, Baschal RM, Gowan K, Tang NL, Alvarado DM, Haller GE, Dobbs MB, Taylor MR, Gurnett CA, **Jones KL**, Miller NH. 2015. Exome sequencing identifies a rare HSPG2 variant associated with familial idiopathic scoliosis. *G3*, Dec 12;5(2):167-74. **PMC4321025**.

3. **RNAseq**: We are also using RNAseq to determine the transcriptional effects of genomic perturbations, and diseases. Current contributions include investigations involving developmental biology in craniofacial development, neural tube closure, retinal development, as well as cancer, immunology and virology.
   a. Dodson RB, Powers KN, Gien J, Rozance PJ, Seedorf G, Astling D, Jones K, Crombleholme TM, Abman SH, Alvira CM. Intrauterine growth restriction decreases NF-κB signaling in fetal pulmonary artery endothelial cells of fetal sheep. American journal of physiology. Lung cellular and molecular physiology. 2018; 315(3):L348-L359. PMID: 29722560 PMCID: **PMC6172617**
   b. Chen Y, Anastassiadis K, Kranz A, Stewart A.F., Arndt K, Waskow C, Yokoyama A, Jones K, Neff T, Lee Y and P Ernst. MLL2, not MLL1, plays a major role in sustaining *MLL*-rearranged Acute Myeloid Leukemia. *Cancer Cell*, 2017 Jun 12;31(6):1-16 PMID: 28609655 PMCID: **PMC5598468**
   c. Van Otterloo E, Feng W, Jones KL, Hynes NE, Clouthier DE, Niswander L, Williams T. MEMO1 drives cranial endochondral ossification and palatogenesis. Dev Biol. 2016 Jul 15;415(2):278-295. doi: 10.1016/j.ydbio.2015.12.024. Epub 2015 Dec 31. PubMed PMID: 26746790; PubMed Central PMCID: **PMC4914435**.
   d. Bradford AP, Jones KL, Kechris K, Chosich J, Montague M, Warren WC, May MC, Al-Safi Z, Kuokkanen S, Appt SE, Polotsky AJ (2015) Joint miRNA/mRNA expression profiling reveals changes consistent with development of dysfunctional corpus luteum after weight gain. *PLoS ONE* 10(8): e0135163 **PMC4530955**

Complete List of Published Work in Bibliography (157 published papers):
https://www.ncbi.nlm.nih.gov/myncbi/18ebvrmKMgokr/bibliography/public/?sortby=pubDate&sdirection=descending

D. Research Support

Ongoing Research Support

BC120183 (Elias) 09/01/13 – 08/31/19
1.2 calendar
BCRP Department of Defense
$555,555
Targeting androgen receptor in breast cancer: enzalutamide as a novel breast cancer therapeutic
The major goal of this project is to confirm whether enzalutamide can block androgen and estrogen driven proliferation of ER+/AR+ breast tumors recently derived from patients, to determine whether enzalutamide will synergize with trastuzumab in vivo in AR+/Her2+ breast cancer cell lines, regardless of ER status, and to determine if MDV3100 inhibits true TNBC but AR+ tumor xenograft growth in vivo.
Role: Co-Investigator

R01 DE024034-01A1 (Artinger) 07/01/15-05/31/20
0.6 calendar
NIH NIDCR
$361,835
Function of Chromatin Modifiers in Cranial Neural Crest Development
The objective of this application is to determine the mechanism by which two families of epigenetic regulators, KAT2a lysine acetyltransferase and PRDM lysine methyltransferases that regulate each other and act to modify the same H3K9 residue on histone 3, function in zebrafish and mouse cNCC development. We will use two excellent developmental model systems and combine genetic tools with live cell imaging of zebrafish and mouse cNCC behaviors and transcriptional studies to tackle the question of why mutations in Kat2a and Prdms lead to craniofacial abnormalities. The overall hypothesis is that these chromatin modifying enzymes act as opposing transcriptional regulators and function cell autonomously to regulate cNCC proliferation and migration.
Role: Co-Investigator

R01 AR068292-01 (Miller) 07/01/15 – 06/30/20 0.3 calendar
NIH NIAMS
$220,000
Familial idiopathic scoliosis: gene discovery and functional studies
Idiopathic scoliosis (IS) is the most common deformity of the spine in children, with an estimated annual cost of over $3 billion per year. Individuals with IS face lifelong issues, including cosmetic deformity, bracing, and surgery, with females at the greatest risk for severe disease. Family history is a known risk factor for IS, but the genetic causes of the disease are not well understood. We will use cutting-edge DNA sequencing to discover new IS genes, shedding much needed light on the disease process, advancing diagnostics, and paving the way for developing novel therapies.
Role: Co-Investigator

1R01 DK032083 (Rewers) 09/01/16 – 06/30/20 0.6 calendar
NIH NIDDK
$250,000
The overarching goal of our research is to develop new biomarkers of islet autoimmunity and to translate these discoveries to prevention of human Type 1A diabetes (T1D). The proposed studies will widen the spectrum of relevant autoantigens and diseases as well as provide opportunities to reduce cost and expand applications.
Role: Co-Investigator

1R01 DK113586-01 (Thurman) 04/01/17 – 03/31/22 0.6 calendar
NIH NIDDK
$225,000
Immunologic Mechanisms of Progressive Glomerulosclerosis

The proposed research is relevant to public health because chronic kidney disease (CKD) is a widespread problem that is associated with significant morbidity and increased mortality. Blood pressure control is currently the primary therapeutic option for treating patients with CKD. The experiments in this proposal will improve our understanding of the mechanisms of CKD progression and may lead to effective new therapies for the treatment of CKD.
Role: Co-Investigator

R03 NS104566-01A1 (Siegenthaler) 09/01/18 – 08/31/20 0.6 calendar
NIH
$155,500
Foxc1 control of meninges formation and function
Here we propose to utilize single-cell transcriptomic analysis in conjunction with meninges defective *Foxc1* mutants to create *unparalleled* molecular characterization of the developing meninges and identify Foxc1-regulated pathways required for meningeal layer formation and function. Data generated here will provide the first complete description of the cellular and molecular makeup of the meninges and provide a critical guide to pathways that drive meningeal development, enabling us to develop new hypotheses that direct future, larger projects.
Role: Co-Investigator

1R01CA224436 (Ernst) 06/01/18-5/31/23 0.6 calendar
NIH NCI
$539,135
MLL Family Histone Methyltransferases in Myeloid Leukemia
The goals of this project are to 1) determine the role of MLL1/MLL2 in AML of distinct cytogenetic categories, 2) to identify pathways regulated by MLL1/MLL2 in adult AML, and 3) to determine the role of H3K4 methylation in collaboration between MLL1 and MLL2.
Role: Co-Investigator

1 R01 CA237608-01 (Foreman) 04/01/19 - 03/31/24 1.2 calendar
NIH NCI
$250,000
Investigation of a Novel Cancer Stem Cell Population in Ependymoma.
It is our conjecture that only through a greater understanding of these ESC will we identify therapeutic targets by which the relapse rate can be reduced in childhood EPN. The specific aims of our research proposal are hypothesis-driven and designed to confirm the existence of, and more fully characterize, the ESC and other EPN tumor subpopulations and identify the cellular lineage hierarchy within the EPN microenvironment, using molecular, histological and functional approaches. Specific aim 1: Confirmation of existence of EPN Stem Cells (ESCs) in pediatric EPN patient samples. Specific aim 2: Determination of in vitro and in vivo tumor initiating properties of EPN stem cells (ESCs) and other EPN tumor subpopulations.

Compeleted Research Support (last 3 years)
3 R01 AR059947-05S1 (Roop) 09/01/17 – 08/31/18
NIH NIAMSD
Testing the Therapeutic Potential of iPS Cells for Inherited Skin Diseases
Epidermolysis bullosa (EB), is a group of rare, incurable, inherited skin blistering diseases that result in severe blistering and scaring, and in some cases, early death. Building on our scientific progress toward the development of a safe, induced pluripotent stem cell (iPSC)-based therapy for EB simplex (EBS), that we achieved during the course of our parent R01, we propose to extend our studies to a more severe type of EB, recessive dystrophic EB (RDEB), and to manufacture clinical grade materials for reprogramming and gene editing for future clinical trials. If the studies outlined in this application result in obtaining approval for a clinical trial for RDEB, these pre-clinical data will pave the way for approval of iPSC-based clinical trials for a variety of other genetic diseases.

1R01HL106209-01A1 Taylor (PI) 04/01/12 – 03/31/17
NIH NHLBI
Whole Genome DNA Sequencing to Identify Novel Genes in Dilated Cardiomyopathy
The major goal of this project is to use whole-exome capture for gene discovery in Dilated Cardiomyopathy

132431 Brzezinski (PI) 07/01/13 - 06/30/16
Boettcher Foundation
Elucidating the mechanisms of cone photoreceptor development and transplantation
The major goal of this project is to determine the mechanisms of cone photoreceptor development using high-throughput sequencing.

1U01DE024429-01 Williams (PI) 05/01/14 – 04/30/19
NIH NIDCR
Facebase 2: craniofacial development and dysmorphology dataset, tool, and resource development
The goal of this application is to study the spatio- temporal gene expression and chromatin signatures of facial rectoderm and mesynchyme development of mouse, wild-type, facial prominences.