Firstname Lastname
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SUMMARY

I am a postdoctoral scientist seeking to advance my career within a biotechnology organization that will utilize my cell biology and oncology knowledge while also fostering professional development. I have 14 years of laboratory research experience with expertise in molecular biology, cancer genomics and translational science. I also excel in project management, team leadership, written communications and public speaking, and am capable of working both independently and as a cross-functional collaborator to efficiently contribute to projects.

KEY SKILLS

Techniques:

In vitro: Cell culture (2D and 3D), multicolor flow cytometry and cell sorting, DNA cloning, CRISPR/Cas9 editing, transfection, virus preparation and viral transduction, DNA/RNA library preparation, single DNA molecule sequencing, WGS, WES, RNA-seq and single-cell RNA-seq, qPCR, ddPCR, ICC, IHC, Western blotting, cell-based microplate assays

In vivo: Orthotopic and subcutaneous tumor implantation, patient-derived xenograft models, genotyping, drug administration (oral, intravenous, intraperitoneal, subcutaneous), intravenous perfusion, *ex vivo* isolation, culture and analysis of cells using FACS, IHC, imaging, scRNA-seq and WGS approaches

Tools and Platforms:

GraphPad Prism, FlowJo, SnapGene, ImageJ, Cell Ranger, Seurat, Loupe Browser, COSMIC, IGV, UCSC Genome Browser, Galaxy, DepMap/CCLE, NCBI GEO, Biorender, Benchling, Microsoft Office Suite (Excel, Word, PowerPoint)

EMPLOYMENT EXPERIENCE

Fourth job

***Postdoctoral Research Fellow*, 11/2023 – 10/2025**

Postdoctoral work studied how innate immune pathway activation, particularly through aberrant DNA-sensing pathways, drives mutagenesis, neoantigen generation and genomic instability in cancer cells, both intrinsically and in response to therapies. I led multiple research projects to identify the causes and consequences of immune activity in breast, lung and colon cancer models, to understand how these processes influence cancer cell evolution and therapy response.

• Curated and analyzed patient datasets (WGS, WES, RNA-seq) to identify mutational processes and candidate driver genes involved in cancer progression and therapy resistance, and to inform subsequent experimental models.
• Designed and generated reporter cell lines via CRISPR-Cas9-mediated homology-directed repair and conducted genome-wide CRISPR screens to identify modulators of drug-induced gene activation
• Optimized and implemented state-of-the-art single DNA molecule sequencing methods to enable detection of rare variants in bulk cell populations
• Trained and supervised interns, MS and PhD students
• Contributed experimental findings to the broader research group by preparation of publications and grant applications

Third job

***PhD Student*, 08/2019 – 10/2023**

Completed doctoral work in the IMAXT laboratory (Imaging and Molecular Annotation of Xenografts and Tumors) in CRUK Cambridge Institute of University of Cambridge. Graduate studies focused on understanding the molecular underpinnings of endothelial differentiation in cancer cells, vasculogenic mimicry and mechanisms of resistance to anti-angiogenic therapies across several solid tumor types.

• Comprehensively characterized the phenotypic and molecular properties of endothelial-like cancer cells capable of vasculogenic mimicry in breast, lung and renal carcinoma models
• Established a method to isolate vasculogenic mimicry-capable cancer cells from multicellular populations using a flow cytometry-based method and validation of this method across a range of tumor types
• Design and generation of a novel CRISPR screening platform, utilizing tRNA-based polycistronic expression of gRNAs, ensuring potent gene knockout and maximum screen efficiency
• Conducted genome-wide CRISPR screens using bespoke libraries to identify drivers of endothelial differentiation and vasculogenic mimicry, identifying potential therapeutic targets
• Designed focused CRISPR library panels compatible with single-cell RNA sequencing technologies and performed deep phenotyping experiments to study transcriptomic effects of gene perturbations
• Generated isogenic knockout and overexpression models of genes of interest and validated both:
• *in vitro* using cell culture assays (3D tube formation, cell viability/survival), RNA-seq and proteomics analysis (IP-MS, RIME)
• *in vivo* using orthotopic models of metastatic breast cancer, combination drug therapy and *ex vivo* analysis using 3D imaging techniques to visualize, differentiate and quantify both tumor-derived and host vasculature, informing rational drug combinations to enhance the efficacy of anti-angiogenic therapies by inhibition of cancer cell endothelial differentiation

Second job

***Associate Scientist*, 01/2016 – 07/2019**

• Designed and executed experiments using both cell culture and mouse models to study the molecular mechanisms of breast cancer metastasis
• Managed the day-to-day workings of the lab to ensure streamlined experimentation
• Analyzed and presented findings at weekly research group meetings, and to the broader community at institute meetings and conferences

First job

***Associate Scientist*, 09/2015 – 12/2015**

• Supported phase II-III clinical trials across oncology and metabolic disease therapeutic areas, ensuring adherence to GCP, SOPs and regulatory guidelines
• Coordinated data collection and quality control to maintain integrity of clinical trial data
• Collaborated with study investigators and project managers to meet milestone timelines

EDUCATION

Doctor of Philosophy, Medical Science

***University of Cambridge,* October 2023**

Thesis: Targeting Vasculogenic Mimicry in Cancer

Bachelor of Science, Biochemistry

***Virginia Commonwealth University,* May 2015**

PUBLICATIONS

. FOXC2 promotes vasculogenic mimicry and resistance to anti-angiogenic therapy. *Cell Reports*, 42(8), 112791.
*Authors contributed equally

Clonal transcriptomics identifies mechanisms of chemoresistance and empowers rational design of combination therapies. *eLife*, 11, e80981.

Addiction of lung cancer cells to GOF p53 is promoted by up-regulation of epidermal growth factor receptor through multiple contacts with p53 transactivation domain and promoter. *Oncotarget*, 7(11), 12426–12446.

p53: its mutations and their impact on transcription. *Subcellular Biochemistry*, 85, 71–90.

Allele specific gain-of-function activity of p53 mutants in lung cancer cells. *Biochemical and Biophysical Research Communications*, 428(1), 6–10.

CONFERENCE PROCEEDINGS

“Induction of APOBEC3 mutagenesis by genotoxic treatments in cancer”. Cancer Genomics Meeting, October 2024
“Impact of APOBEC3 mutagenesis on therapy resistance in cancer”. Cancer Center Retreat, June 2024

AWARDS AND LEADERSHIP

• Finalist, Cancer Research Thesis Prize, 2023
• President, Cancer Research Graduate Society, 2019-2020
• Innovation and Discovery Award, 2018