Dr. Sarah Bowling is an Assistant Professor in the Department of Developmental Biology at Stanford University School of Medicine, and is an Affiliate Member of the BASE Initiative. Sarah carried out her PhD at Imperial College London, where her work focused on understanding the mechanisms and roles of cell competition during early mammalian development. For her postdoctoral research, Sarah moved to Boston Children's Hospital and the Harvard Department of Stem Cell and Regenerative Biology. Here, she co-developed new lineage tracing mouse models that enable the simultaneous tracing of thousands of cells in vivo with unique, transcribed cellular barcodes.

Sarah joined Stanford University in 2024. Her research focuses on understanding lineage formation and tissue growth in mammalian development during normal and perturbed embryogenesis. Her laboratory uses a combination of next-generation tools and classical embryological approaches to uncover mechanisms of plasticity and resilience during mammalian embryo development, with the aim of using this knowledge to extend our understanding of regeneration and developmental diseases.

ACADEMIC APPOINTMENTS

• Assistant Professor, Developmental Biology
• Member, Bio-X

LINKS

• Bowling Lab Website: bowlinglab.org

Research & Scholarship

CURRENT RESEARCH AND SCHOLARLY INTERESTS

The Bowling lab focuses on understanding lineage formation and tissue growth in mammalian development during normal and perturbed embryogenesis. We use a combination of next-generation tools and classical embryological approaches to uncover mechanisms of plasticity and resilience during mammalian embryo development, with the aim of using this knowledge to extend our understanding of regeneration and developmental diseases.

Teaching

STANFORD ADVISEES

Postdoctoral Faculty Sponsor

Marine Secchi
Publications

PUBLICATIONS

- Mutation of p53 increases the competitive ability of pluripotent stem cells. *Development (Cambridge, England)*
  2024; 151 (2)

- A mouse model with high clonal barcode diversity for joint lineage, transcriptomic, and epigenomic profiling in single cells. *Cell*
  2023; 186 (23): 5183-5199.e22

- DRP1 levels determine the apoptotic threshold during embryonic differentiation through a mitophagy-dependent mechanism. *Developmental cell*
  2022; 57 (11): 1316-1330.e7

- Lifelong multilineage contribution by embryonic-born blood progenitors. *Nature*
  2022; 606 (7915): 747-753

- Cell competition acts as a purifying selection to eliminate cells with mitochondrial defects during early mouse development. *Nature metabolism*
  2021; 3 (8): 1091-1108

- An Engineered CRISPR-Cas9 Mouse Line for Simultaneous Readout of Lineage Histories and Gene Expression Profiles in Single Cells. *Cell*
  2020; 181 (6): 1410-1422.e27

- Genetic Deletion of Hex1 Promotes Exit from the Pluripotent State and Impairs Developmental Diapause. *Stem cell reports*
  2019; 13 (6): 970-979

  Bowling, S., Lawlor, K., Rodriguez, T. A.
  2019; 146 (13)

- P53 and mTOR signalling determine fitness selection through cell competition during early mouse embryonic development. *Nature communications*
  2018; 9 (1): 1763

- Cell Competition and Its Role in the Regulation of Cell Fitness from Development to Cancer. *Developmental cell*
  Di Gregorio, A., Bowling, S., Rodriguez, T. A.
  2016; 38 (6): 621-34