Ann Richmond

Ann received her PhD from Emory University in 1979 and did postdoctoral work at Emory University School of Medicine. In 1983, she was appointed Assistant Professor of Medicine at Emory where her research focused on the identification and characterization of melanoma growth stimulatory activity (MGSA), later named CXCL1. After determining the cDNA sequence, chromosomal location for MGSA, and sequence of the gene with its promoter, her group continued to characterize its function in tumorigenesis and leukocyte trafficking. In 1989 she moved to Vanderbilt University as Associate Professor of Cell Biology with tenure, and later affiliated with the Department of Cancer Biology where from 2000-2018 she served as Vice Chair of the Department of Cancer Biology, followed by serving as Director of the Program in Cancer Biology from 2018-2024 and as Professor of Pharmacology. She is a fellow of the ELAM program for women in leadership, served as the Vanderbilt University Assistant Dean of Postdoctoral Affairs from 2005-2010, as Associate Director of Education for the Vanderbilt-Ingram Cancer Center from 2004-2020.

At Vanderbilt, her lab characterized the functions and regulation of CXCR2, the chemokine receptor for MGSA/ CXCL1 and demonstrated that CXCR2 plays a major role in angiogenesis, wound healing, tumor growth, inflammation, and recruitment of immunosuppressive myeloid cells into tumors. They also showed how phosphorylation of the serine and threonine residues in the C-terminal domain of the receptor plays a key role in downregulation of signaling/receptor trafficking and identified key proteins (VASP, AP2, PP2A, HIP, LASP,and others) that associate with the carboxy-terminal domain of CXCR2 to maximize its activity. Her group also showed that NF-κB is a major regulator of transcription of the CXC-chemokines and that inhibition of NF-κB in tumor cells can inhibit tumor growth, while inhibition NF-κB in myeloid cells shifts the phenotype of macrophages in the tumor microenvironment (TME) to an immunosuppressive phenotype and enhances tumor growth. Moreover, targeted deletion of CXCR2 in myeloid cells altered the tumor immune microenvironment and inhibited tumor growth in part through reducing MDSC recruitment to the TME. Additionally, targeted deletion of CXCR2 during melanocyte differentiation transcriptionally reprogramed the tumor microenvironment and reduced both tumor formation and growth. Her team has shown how combining immune checkpoint inhibitors (ICI) with therapies that target CXCR1/CXCR2, the PI3K/AKT pathway, or the RAS/RAF/PI3K improves inhibition of tumor growth. This work led to the development of new clinical trials combining CXCR/CXCR2 inhibitors, or RAS/RAF/PI3K inhibitors with ICI for treatment of cancers. Her work has been continuously funded by grants from the NCI and the Department of Veterans Affairs since 1983, in addition to other foundations and societies.

Richmond received the Associate Career Scientist Award from the VA (1988-2024) and an endowed Ingram Professor of Cancer Research Award (2005-2025). She received the Charles R. Park Research Award in 2014, the William S. Middleton Award for Excellence in Biomedical Laboratory Research in 2016, was named Fellow in the AAAS in 2018, received the Delores Shockley Partnership Award in 2018, and the Legacy Award from the Society for Leukocyte Biology in 2019. Ann has been active in many professional societies, including the SLB, as council member (1999-2002), as president elect (2012-2014), and as president (2014-2016). Since 1995 she has served on the JLB editorial board where she was also section editor. She has published over 220 peer reviewed papers, chapters, and review articles, trained over 70 undergraduate, graduate and postdoctoral fellows, and has been a champion for advancing training in cancer research in diverse populations.