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Biosketch

Dr. Venters earned his PhD in Molecular Biology from Pennsylvania State University where he studied epigenetic and gene regulatory mechanisms. During his postdoctoral training at Penn State, he developed state-of-the-art functional genomic tools that enable examination of protein-DNA interactions with the highest resolution currently possible. Research in the Venters lab involves (1) development of cutting edge functional genomic and computational tools, (2) application of these tools to dissect fundamental erythroid cell biology, and (3) translation of key discoveries into new therapeutics and treatment strategies.

Key Publications

"Integrative view of epigenetics in erythroid cells," 2018, *Current Opinion in Hematology*, 25(3):189-195.

"Epo reprograms the epigenome of erythroid cells," 2017, *Experimental Hematology*, S0301-472X(17)30133-9.

"Genomic Organization of Human Transcription Initiation Complexes," 2016, *PLoS ONE*, 11(2):e0149339.

"How hormones impact red blood cells in health and disease"

The Venters lab leverages their expertise in Molecular Biology, Functional Genomics, and Bioinformatics to **study the molecular mechanisms underlying hematological diseases**. Our research is guided by two overarching questions:

1. How does EPO signaling control RNA polymerase II kinetics during red cell development?
2. How are early EPO-responsive genes regulated by transcription factors and enhancers?

Erythropoietin (EPO) is the primary hormone regulator that controls erythroid cell maturation, a process that is required for the **daily replenishment of nearly 1% (200 billion) of the circulating red blood cells**. Recombinant human EPO (trade name Epogen) is a \$5 billion/year drug that is used to treat chronic anemia. However, the therapeutic use of EPO has been controversial since reports have emerged of its link to **increased risk of heart attacks, tumor growth, and death in some cases**. An understanding of EPO function has advanced substantially, but **certain aspects of the EPO signaling pathway remain unknown**. In particular, how EPO signaling controls erythroid expression patterns through epigenetic and transcriptional mechanisms remains poorly understood.

Thus, the overarching goal of our research is to **understand the gene regulatory mechanisms governing EPO-induced erythroid differentiation, and translating these discoveries into potential therapeutic interventions**.

