

**From:** Inform  
**Subject:** EM: Dept. of Natural Sciences Seminar Announcement - 11/07

**From:** Lauran Soto

## **DNS SEMINAR ANNOUNCEMENT:**

### ***ING4 at the intersection of stem cell fate, immune cell differentiation, and tumorigenesis***

Dr. Katie Kathrein  
Assistant Professor  
Department of Biological Sciences  
University of South Carolina

Friday, Nov. 7th, 2025  
12:15p-1:15p  
Burns Lecture Hall - NS E007  
(Overflow viewing in E050)

#### **Abstract:**

Defining the mechanisms that regulate how stem cells balance quiescence, self-renewal, and differentiation is critical for understanding both normal development and disease. The chromatin remodeling factor and tumor suppressor, Inhibitor of Growth 4 (ING4), plays a key role in this balance. Using mouse, zebrafish, and cell-based model systems, we found that ING4 orchestrates the molecular networks that control hematopoietic stem cells (HSCs), which supply all the immune cells required by an organism. Loss of ING4 leads to altered immune cell differentiation and function, including the development of bone marrow failure in the red blood cell compartment, causing anemia. Bone marrow failure often occurs through prolonged activation of HSCs, ultimately leading to exhaustion and loss of HSC function. Surprisingly, in the absence of ING4, HSCs exhibit transcriptional signatures of activation through expression of stress-response genes, yet they remain phenotypically quiescent and functional. The role of ING4 in HSCs highlights the fine balance between HSC readiness for stress response and maintenance of long-term dormancy. As these same pathways are often aberrantly expressed in cancer, our work suggests that shared molecular programs contribute to stem cell dysregulation and tumorigenesis in the absence of ING4. Supporting this, inhibition of the key immune response regulator, NF- $\kappa$ B, suppresses tumor growth in ING4-deficient cancer models. Together, these findings position ING4 as a critical regulator of stem cell fate and immune cell differentiation, revealing how disruption of this balance can drive disease.



Department of Natural Sciences  
Seminar Series

***ING4 at the intersection of stem cell  
fate, immune cell differentiation, and  
tumorigenesis***



Dr. Katie Katherin  
Assistant Professor  
Department of Biological Sciences  
University of South Carolina  
**November 7, 2025| 12:15-1:15 PM**  
**Nucleus E007 (Burns Lecture Hall)**

Defining the mechanisms that regulate how stem cells balance quiescence, self-renewal, and differentiation is critical for understanding both normal development and disease. The chromatin remodeling factor and tumor suppressor, Inhibitor of Growth 4 (ING4), plays a key role in this balance. Using mouse, zebrafish, and cell-based model systems, we found that ING4 orchestrates the molecular networks that control hematopoietic stem cells (HSCs), which supply all the immune cells required by an organism. Loss of ING4 leads to altered immune cell differentiation and function, including the development of bone marrow failure in the red blood cell compartment, causing anemia. Bone marrow failure often occurs through prolonged activation of HSCs, ultimately leading to exhaustion and loss of HSC function. Surprisingly, in the absence of ING4, HSCs exhibit transcriptional signatures of activation through expression of stress-response genes, yet they remain phenotypically quiescent and functional. The role of ING4 in HSCs highlights the fine balance between HSC readiness for stress response and maintenance of long-term dormancy. As these same pathways are often aberrantly expressed in cancer, our work suggests that shared molecular programs contribute to stem cell dysregulation and tumorigenesis in the absence of ING4. Supporting this, inhibition of the key immune response regulator, NF- $\kappa$ B, suppresses tumor growth in ING4-deficient cancer models. Together, these findings position ING4 as a critical regulator of stem cell fate and immune cell differentiation, revealing how disruption of this balance can drive disease.

For more information, contact Pete Chandrangsu (pchandrangsu@natsci.claremont.edu )

Best,  
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