The Leukemia and Lymphoma society of Canada reports that one Canadian will be diagnosed with a hematological malignancy every 28 minutes, and another will die every 77 minutes. It is vital to understand the mechanisms involved in order to discover new therapeutic avenues. Polo-like kinase 4 (PLK4) is a serine/threonine kinase that is critical for proper centrosome duplication. A loss of Plk4 is associated with mitotic failure and plk4<sup>+/−</sup> cells display genomic instability. In human patients with leukemia, lymphoma, and MDS, the plk4 promoter was found to be hypermethylated resulting in lower protein expression in these malignancies. Therefore PLK4 may play an important role in the cell that implicates it in hematopoiesis. The plk4 heterozygous (plk4<sup>+/−</sup>) mouse may represent a novel model of hematological malignancy. These mice develop splenomegaly (enlarged spleen) at an incidence of 30% compared to 7% in their wildtype littermates. Enlarged spleen histology reveals a distinct loss of cellular organization and compartmentalization of the red and white pulp of the tissue. Our plk4<sup>+/−</sup> mice also show a change in circulating white blood cells (WBC), and those with splenomegaly display striking hypercellular bone marrow histology. This phenotype is indicative of a hematological malignancy, however it could represent a very broad spectrum of disorders. Hematopoietic cancers can be categorized as either being myeloid or lymphoid in origin. Flow cytometry experiments and hematology counts in aged mice are suggesting a myeloid neoplasm at this time. This research is significant as it will validate a novel mouse model and work to understand the cellular mechanisms potentially driving this phenotype.