Inflammation of the heart, myocarditis, is a leading cause of sudden death from heart failure in children and adults under age 50 both domestically and internationally. Myocarditis, often caused by an infection, can progress to dilated cardiomyopathy and chronic heart failure, often warranting a heart transplant. A large gap in knowledge in the study of myocarditis includes unknown mechanisms of disease progression and immune activation as well as limited diagnostic biomarkers. This gap is then reflected in clinical care where there is little to no intervention or treatment available for patients with myocarditis. Therefore, it is critical to better understand the mechanisms associated with disease progression. Previous literature has discovered that vitamin D binding protein (DBP) is upregulated in patients with myocarditis, more so in men than women. DBP is implicated in many inflammatory diseases and is thought to be involved in the tissue injury response by acting as a chemotactic cofactor to enhance neutrophil recruitment. Compared to DBP deficient mice (DBP-/-), we have found that inflammation is greater in male and female wildtype (DBP+/+) mice at both day 5 post-infection (d5pi) and day 10 post-infection (d10pi). Giving infected mice recombinant DBP resulted in higher inflammation when compared with infected mice given saline. Furthermore, we have discovered that DBP-/- mice naturally have less neutrophils than DBP+/+ mice at both timepoints of infection, indicating a link between DBP and neutrophils. Utilizing 1A8 anti-Ly6g antibody and its isotype, 2A3, we found that DBP+/+ mice depleted of neutrophils experience less inflammation, contributing to the idea that neutrophil chemotaxis plays a big role in the development of myocarditis. We have also found that neutrophil chemotaxis is greater in DBP+/+ infected males than DBP+/+ uninfected males and DBP-/- infected males. These data indicate that DBP and neutrophils contribute to myocarditis and reinforce a connection between DBP and neutrophils in instigating inflammation. This preliminary work is expected to provide a mechanistic understanding of the role of circulating DBP and neutrophils in myocarditis severity, potentially making them feasible therapeutic targets.