BRCA1 and PALB2 are two proteins that bind to efficiently repair DNA damage through homologous recombination. Inability for these proteins to dimerize due to genetic variations can increase an individual's risk of developing breast and ovarian cancer. Currently, most PALB2 genetic variants are classified as variants of unknown significance (VUSs) due to insufficient data to predict pathogenicity. *In vivo* methods to predict pathogenicity of these variants are time consuming and costly. As a result, we aimed to create a high-throughput and cell-free assay to test the effect of VUSs on the BRCA1-PALB2 binding interaction. Importantly, we wanted to recreate any relevant cellular conditions to obtain the most accurate data, and currently, the effect of PALB2 phosphorylation on the BRCA1-PALB2 binding interaction *in vitro* is unknown. To determine if phosphorylation affects the binding interaction, we mimic the phosphorylation states of PALB2 using site-directed mutagenesis and test their effect on BRCA1 binding using isothermal titration calorimetry. Our results indicate a surprising finding: PALB2 phosphorylation does not significantly alter the strength of the BRCA1-PALB2 binding interaction with minimized constructs *in vitro*. Thus, we hypothesize it is not critical to recreate the phosphorylation states of PALB2 when testing the effect of VUSs on the BRCA1-PALB2 binding interaction.