Schizophrenia is a severe psychiatric disorder characterized—among many other symptoms—by hallucinations, delusions, and cognitive impairments. The hippocampus, a brain region involved in learning and memory, displays abnormal neurobiology in patients with schizophrenia, and this aberration contributes to psychosis. Excitatory glutamatergic neurons comprise 90% of hippocampal neurons; the remaining subsets are inhibitory GABAergic interneurons that synapse locally onto excitatory neurons. Studies show disinhibition of glutamatergic neurons in patients with schizophrenia and a decreased number or activity of parvalbumin interneurons, one of the largest groups of inhibitory interneurons within the hippocampus. Thus, this study analyzes the optical density and total neuronal density of parvalbumin interneurons in human postmortem tissue. We tested the hypothesis that there is a decreased neuropil or neuronal density of Parvalbumin and/or a decreased number of parvalbumin immunolabeled interneurons. Using post-mortem tissue from the Alabama and Maryland Brain Collections, the hippocampus was sampled in five schizophrenia cases and six matched controls. Parvalbumin protein was detected and analyzed using immunohistochemistry at the light microscopic level on Image J software. We found no differences between groups for any of the three measures. We found preliminary significant correlations between race and PV labeled neuropil level and PV positive neurons between groups. Our investigation was limited due to our small cohort. Future directions include analyzing parvalbumin tissue at the electron microscopic level, as it offers the opportunity to visualize the subcellular localization of specific abnormalities, such as decreases in synaptic density and mitochondrial structural integrity, in a layer-specific way.