Reactive oxygen species (ROS) can react with DNA and lead to damage if ROS are overproduced beyond the beneficial level. To combat the deleterious effect on DNA, cells have a range of repair pathways in their arsenal—base excision repair (BER) being one of them. MUTYH is an adenine glycosylase that is part of the BER pathway. This enzyme recognizes the 8-oxoG:A lesion in DNA that is formed when guanine is oxidized to 8-oxo-7,8-dihydroguanine, which can mispair with adenine. Defective MUTYH have been linked to the hereditary condition, MUTYH-associated polyposis (MAP), that results in the patient developing multiple adenomatous colon polyps, which increases their risk for colorectal cancer significantly. Over 300 MUTYH variants have been found and several of these variants have been linked to MAP. In order for MUTYH to properly bind DNA and have active glycosylase activity, it needs an iron-sulfur cluster present as a cofactor. Since the iron-sulfur cluster is important to the enzyme's functionality, investigating the biological effects and chemical aspects of several MAP variants that surround the cluster is the goal of this research. The kinetic parameters and mutation suppressing functionality of the enzyme variants will be analyzed and compared to the wild type.