Steroid hormones are a class of compounds that play a role in regulating many functions. Glucocorticoid hormone is a compound in this class, which helps maintain homeostasis, including regulation of production of γ-fibrinogen, a protein that plays a major role in blood clotting. Glucocorticoid hormone acts by binding to an intracellular receptor protein called the glucocorticoid receptor (GR). GR bound to glucocorticoid then moves to the nucleus, where it interacts with another protein, *Xenopus* Glucocorticoid Receptor Accessory Factor (XGRAF), to form a heterodimer. This heterodimer binds to an upstream regulatory region of the DNA coding for the γ-fibrinogen gene that is composed of a binding site for XGRAF adjacent to a half GR recognition site (a classical GR response element consists of two elements). Binding of this heterodimer to the recognition sites regulates transcription of the γ-fibrinogen gene. The dimerization interaction relies on specific amino acid sequences on both proteins. These experiments will examine the amino acids on GR that are involved in heterodimerization with XGRAF. Androgen receptor (AR), is very similar to GR, so examining the differences in binding in the presence of XGRAF due to the substitutions could help determine what regions of GR are essential to XGRAF binding. To study the heterodimerization, several constructs that incorporate AR at different parts of GR in place of the normal sequence were expressed in a bacterial system and then isolated for analysis. The proteins were used in gel mobility shift assays which allow detection of the interaction between the nuclear receptors and XGRAF. After studying the binding of these constructs we have determined that the parts of AR that were substituted for GR still allow heterodimerization with XGRAF. Since both GR and AR have the ability to interact with XGRAF, we can speculate that similar types of heterodimerization mechanisms for nuclear receptors could be more common than previously thought.