In the human body, the thyroid system is responsible for influencing functions ranging from neurodevelopment and growth to metabolic regulation. The pituitary hormone thyrotropin (TSH) stimulates the thyroid gland to produce thyroid hormones (TH) which feedback to the pituitary to regulate TSH production to maintain homeostasis or steady-state TH levels. TH regulate protein synthesis, gene expression in every tissue in the body primarily via the thyroid hormone-dependant transcriptional regulation of a gene through the thyroid hormone receptor DNA binding protein (TR) and the proteins that it recruits. Recent research has indicated that the common xenobiotic endocrine disruptor found in many plastics, bisphenol a (BPA), has the ability to mimic TH and thereby disrupt the thyroid system. While preliminary data points to aberrant TR functioning in the chemical's presence, little is known about the actual sub-cellular mechanisms that are altered in the presence of toxic BPA concentrations. This study attempts to clarify such affected mechanisms by observing the toxin's effect on both TR localization and the protein recruitment profile of the TR on the negatively regulated thyroid stimulating hormone (TSH) gene. By using western blots in conjunction with electrophoretic mobility shift assays, streptavidin-agarose pulldowns, and mass spectrometry, we were able to confirm BPA's effects on the TR by identifying specifically recruited transcriptional cofactors whose presence is altered in cells grown in a $1x10^{-6}$ BPA concentration (when compared with controls). Such results not only clarify BPA's harmful effects at a sub-cellular level, but also identify possible target proteins that many of interest in future functional analyses.