Extensive research has repeatedly demonstrated the ability of RNA binding proteins (RBPs) to exhibit control over cellular phenotypes as well as have vital roles in health and disease states. In recent years, RBPs have emerged as critical regulators of the cardiovascular system, as well as key mediators of cardiovascular disease (CVD) development, the leading cause of mortality worldwide. Advances in regenerative medicine, largely the use of induced pluripotent stem cells, have provided powerful tools to study vascular health and deepen our understanding of molecular mechanisms of endothelial cell dysfunction. Previously, we found QKI-7 to be significantly upregulated in diabetic endothelial cells, due to dysregulation of RNA splicing factors CUG-BP and hnRNPM, and greatly impair endothelial cell barrier, compromise angiogenesis and enhance monocyte adhesion. Furthermore, knockdown of QKI-7 in vivo, using a hindlimb ischemia diabetic mouse model, resulted in significant reperfusion and blood flow recovery. Elucidation of QKI-7 as a regulator of endothelial health therefore has substantial potential to reveal novel therapeutic strategies for diabetic individuals. To evaluate this hypothesis and identify enriched RNAs as a result of QKI-7 overexpression, RNA-immunoprecipitation Sequencing was performed on induced pluripotent stem cell derived endothelial cells (iPSCs). Differential expression analysis was performed using the R package DESeq2, followed by standard visualization and interpretation using other R packages including EnhancedVolcano and Pheatmap. Comparative enriched RNAs were filtered according to the absolute log2 fold change greater than +1 and corrected p-value threshold of <0.05. Of a total of 2129 differentially enriched RNAs, we isolated 42 RNAs (17 Coding and 25 Non-Coding) to be statistically significantly enriched by QKI-7. To allow for the elucidation of the potential pathogenic mechanisms, Ingenuity Pathways Analysis was performed to highlight canonical pathways affected by QKI-7 overexpression. The enriched RNAs were revealed to have fundamental roles in both the cardiovascular system development and CVD development networks as well as be affiliated with 48 pathways connected to vasculature health including processes such as vasculogenesis, angiogenesis, and endothelial cell growth and proliferation. Encouragingly, many were shown to have an affiliation with diabetes and diabetic complications also. Overexpression of QKI-7 in ECs confirmed QKI-7 to significantly regulate the expression of 20 of these enriched RNAs. Moreover, using the platform RBPmap, numerous binding sites of QKI-7 within these enriched RNAs were identified. Through direct gene regulation, the RBP QKI-7 is able to bind and mediate a vast network of endothelial cell dysfunction, contributing to the progression of CVD in diabetic individuals. Manipulation of QKI-7 therefore represents a promising strategy for the treatment of diabetic vascular complications.