

Public Abstract

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Title:INVESTIGATION OF THE BIOLOGICAL ROLE OF THE POLYCYSTIC KIDNEY DISEASE PROTEIN BICAUDAL C (BICC1) USING COMPARATIVE ANIMALS MODELS

Polycystic kidney disease (PKD) is the most common life-threatening genetic disorder in humans, affecting 600,000 Americans and more than 12 million people worldwide. PKD is more common than cystic fibrosis, muscular dystrophy, Down's syndrome, sickle cell anemia, and hemophilia combined. PKD is characterized by bilateral renal cysts that grow and multiply over time, impeding kidney function and ultimately leading to renal failure. PKD is the principal cause of end-stage renal disease in adults and children. Currently there is no cure for PKD and treatment is limited to costly dialysis and organ transplantation. Although many rodent models have been established that mimic certain aspects of human PKD and have been useful in the identification of disease-causing genes, the specific molecular mechanisms involved in cyst formation remain unclear. Bicaudal-C (Bicc1) is the disease-causing gene in the juvenile congenital polycystic kidney (jcpk) mouse model for PKD. The function of Bicc1 is unknown; however the Bicc1 protein contains two conserved regions, K-homology (KH) regions which interact with RNA and a sterile alpha motif (SAM) region which interacts with other proteins. We hypothesize that Bicc1 plays an integral role in normal kidney development. The objective of the current research was to investigate the biological role of Bicc1 in the kidney using comparative animal models. In this study, we investigated the RNA and protein interactions of the Bicc1 protein and established an alternative, comparative zebrafish model with which to further study the molecular function of Bicc1 in living organisms. Further examination of these interactions will lead to an increased understanding of the underlying molecular mechanisms involved in cyst formation and hopefully bring us closer to a cure.