Epilepsy is the main negative factor affecting mental health. The estimated proportion of the general population with active epilepsy is 4-10 per 1000 people. Globally, an estimated 2.4 million people are diagnosed with epilepsy each year. There are many factors causing epilepsy. To understand how epilepsy develops, the studies of the molecular mechanisms of hereditary forms of epilepsy could be instrumental. One of these is early infantile epileptic encephalopathy (EIEE, Ohtahara et al., 1976). Its incidence is 1/100 000 births in Japan and 1/50,000 births in the UK. Currently, more than 70 different variants of this disease have been found linked to mutations in different genes (http://omim.org/entry/308350#references), including the voltage-gated ion channels Kv2.1/KCNB1 (EIEE26). There is a significant phenotype heterogeneity, which is difficult to categorize due to the limited pool of specimens. This task is complicated because mutations in this gene result in different clinical outcomes, including epileptic encephalopathy (i.e., mental degeneration following the onset of epileptic seizures), autism, verbal deficit (de Kovel et al., 2017; Torkamani et al., 2014). In such cases, the study in animal systems such a developing zebrafish, where analysis of a large number of specimens is not a problem, may help understand the cause of disease at the molecular level. Further, it may help develop a treatment in the form of a drug to cure or relieve acquired epilepsy. Therefore, we developed the zebrafish-based test system based on the analysis of defects in the developing mutant zebrafish deficient in Kcnb1. We plan to study the effect of different mutant forms of KCNB1 identified in humans on the development of the brain and sensory organs in zebrafish. The analysis results will help understand the molecular mechanisms of epilepsy, define different categories of mutations, and develop reliable therapy for human patients.