The SCN1A gene belongs to a family of genes that provide instructions for making sodium channels. ... The SCN1A gene provides instructions for making one part (the alpha subunit) of a sodium channel called NaV1.1. These channels are primarily found in the brain, where they control the flow of sodium ions into cells.

The absence of studious seizure in SCN1A mutation-positive babies for the first 6 months raises the possibility that the consequences of mutation in SCN1A are subsidized by EGRs from BML. EGR-dependent-modifier gene effect is likely imposed by the other members of the SCN family. Therefore, we advocate that miRNA/lncRNA from BML and bacosides/miRNA from BMI buffer the effect of SCN1A mutation by sustainably maintaining modifier gene effect in the aberrant neurons. The presence of miRNA-155-5p, -30b-5p, and -30c-5p family in BML and miR857, miR168, miR156, and miR158 in BMI target at regulating SCN family and CLCN5 as visualized by Cystoscope. Thus, we envisage that the possible effects of EGR might include (a) upregulating the haploinsufficient SCN1A strand, (b) down-regulating seizure-elevated miRNA, © suppressing the seizure-induced methyltransferases, and (d) enhancing the GluN2A subunit of NMDA receptor to improve cognition. The potential of these EGRs from BML and BML is to further experimentally strengthen, long-haul step forward in molecular therapeutics [1)].

1)
Suvekbala V, Ramachandran H, Veluchamy A, Mascarenhas MAB, Ramprasath T, Nair MKC, Garikipati VNS, Gundamaraju R, Subbiah R. The Promising Epigenetic Regulators for Refractory Epilepsy: An Adventurous Road Ahead. Neuromolecular Med. 2022 Sep 24. doi: 10.1007/s12017-022-08723-0. Epub ahead of print. PMID: 36153432.