Alcohol use disorders (AUDs) are a major public health issue and produce enormous societal and economic burdens. Current Food and Drug Administration (FDA)-approved pharmacotherapies for treating AUDs suffer from deleterious side effects and are only effective in a subset of individuals. It is therefore essential to find improved medications for the management of AUDs. Emerging evidence suggests that anticonvulsants are a promising class of drugs for treating individuals with AUDs. This study has used an integrative functional genomics approach to demonstrate that genes that encode Kv7 channels (i.e. Kcnq2/3) are related to alcohol (ethanol) consumption, preference and acceptance in rodents. Retigabine (FDA-approved anticonvulsant), a novel Kv7 channel opener, was shown reduce voluntary ethanol consumption of Wistar rats in a two-bottle choice intermittent alcohol access paradigm. Systemic administration and microinjections of retigabine into the nucleus accumbens significantly reduced alcohol drinking, and retigabine was more effective at reducing intake in high- versus low-drinking populations of Wistar rats. Prolonged voluntary drinking increased the sensitivity to the proconvulsant effects of pharmacological blockade of Kv7 channels and altered surface trafficking and SUMOylation patterns of Kv7.2 channels in the nucleus accumbens. These data implicate Kcnq2/3 in the regulation of ethanol drinking and demonstrate that long-term drinking produces neuroadaptations in Kv7 channels. In addition, these results have identified retigabine as a potential pharmacotherapy for treating AUDs and Kv7 channels as a novel therapeutic target for reducing heavy drinking.