1128-92-225 **Anatoly AB Buchin*** (bigneuron@alleninstitute.org), 615 Westlake Ave N, Seattle, WA 98109, **Anton AC Chizhov** (anton.chizhov@mail.ioffe.ru), 26 Politekhnicheskaya, Saint Petersburg, St Petersb 194021, **Gilles GH Huberfeld** (gilles.huberfeld@mac.com), 47-83 Boulevard de l'Hôpital, 75013 Paris, Ile de Fra, France, **Richard RM Miles** (richard.miles@upmc.fr), 47 Boulevard Hôpital, 75005 Paris, Ile de Fra, France, and **Boris BG Gutkin** (anat.buchin@gmail.com), 29 rue d'Ulm, 75005 Paris, Ile de Fra, France. *Reduced efficacy of the KCC2 cotransporter promotes epileptic oscillations in a subiculum network model.*

Ion regulation in the brain is a major determinant of neural excitability. Intracellular chloride in neurons, partial determinant of the resting potential and the inhibitory reversal potentials, is regulated together with extracellular potassium via kation chloride cotransporters. During temporal lobe epilepsy the homeostatic regulation of intracellular chloride is impaired in pyramidal cells, yet how this dysregulation may lead to seizures has been unexplored. Using realistic neural network model describing ion mechanisms we show that chloride homeostasis pathology provokes seizure activity analogous to recordings from epileptogenic brain tissue. We show that there is a critical percentage of pathological cells required for seizure initiation. Our model predicts that restoration of the chloride homeostasis in pyramidal cells could be a viable anti-epileptic strategy. (Received February 27, 2017)