Tuberous Sclerosis Complex (TSC)

Pre-Questions

1. Describe the important problem or critical barrier that impedes progress in this field.

-The critical barrier that was found is that the Kv1.1 voltage gated potassium channel protein in neurons was expressed on dendrites. This caused the mTOR kinase to be inhibited and it is very important for neuronal signaling. Rapamycin increased KV1.1 and its local synthesis needs to be reduced. The synaptic excitation may be able to reduce the local synthesis of Kv1.1 channels.

1. Write 3 questions to ask the speaker based on your pre-lecture reading?
* What exactly does Kv1.1 channels do to the body that it needs to be reduced?
* How much rapamycin was given when it caused an increase in the Kv1.1 channels?
* What is the difference in Kv1.1 and Kv1.4? Why did rapamycin increase kv1.1, but not Kv1.4 in the CA1 dendritic field?

Answer 4 of the following questions:

1. What may be the benefits of the research to society?

-The benefit of this research is that may possibly be the cure to other forms of TSC if it is perfected. That is a huge benefit to the world, to find one cure for several diseases.

1. How will the successful completion of the goals of this research advance scientific knowledge, technical capability, and/or clinical practice be improved?

Since synaptic excitation leads to a decrease of Kv1.1 channels, it can possibly lead to a cure for Tuberous Sclerosis Complex (TSC).

1. Are the concepts, approaches or methodologies, instrumentation, or interventions novel to one field of research or novel in a broad sense?

The concepts, approaches or methodologies are novel in abroad sense because it is covering more than one field. If perfected it can treat several different disorders.

1. 4. Is a refinement, improvement, or new application of theoretical concepts, approaches or methodologies, instrumentation, or interventions proposed?

A new application of theoretical concepts and approaches has been proposed. They localized endogenous Kv1.1 mRNA to dendrites and then distinguished Kv1.1 synthesized in soma and transported to dendrites. After the growing of Kaede Kv1.1 they explored how Kv1.1 local translation could be regulated.

Post

This article is about a study on Tuberous Sclerosis Complex (TSC). TSC is a rare multi-system disorder, manifestations of benign tumors and lesions in several organs of the body. People with this disorder usually suffer from anxiety, depression, epilepsy, and ADHD. TSC is a caused by the mutations in the Tsc1and Tsc2 genes. These genes encode for hamartin and tuberin and they start a process that leads to the inhibition of mTORC1.

 In order to treat this disease a drug needs to be found that can work jointly with rapamycin. Rapamycin works great in curbing the epilepsy and the over growth of tumors. However, it does not eliminate them. This drug needs to block autophagy and increase apoptosis. It also needs to take over the metabolism of TSC1 and TSC2 depleted cellsto increase apoptosis. Also, the reactive oxygen needs too be corrected because it disrupts TSC cells. Although these ideas are proposed, cell biology of the TSC cells need to be studied more.