***Response to Sundaram and Garg***

In Rao et al., 2018, we found that the Golgi-apparatus localizes to the primary dendrite early in adult-born DGC development. Here, Sundaram and Garg discuss previous findings in *Drosophila* DA neurons demonstrating the importance of dendritic Golgi outposts found at dendritic branch-points, in acentrosomal microtubule nucleation, and its role in dendrite branching and maturation (Ori-McKenney, Jan et al. 2012). We observed that the Golgi extended into the DGC primary dendrite by 14 days, however, the presence of dendritic Golgi outposts in DGCs, or the role of the Golgi-complex in DGC dendrite branching and maturation, were not determined in our study. It is possible that dendritic Golgi outposts in DGCs were not detected using the cis-Golgi marker, GRASP65. The presence and role of Golgi outposts in dendrite establishment of adult-born DGCs, thus remain for future investigation.

Sundaram and Garg further discuss our findings on the involvement of the STK25 Golgi-associated complex in DGC dendrite establishment, and compare these findings to our previous study on the role of the LKB1-STRAD complex in axon specification in the embryonic cortex (Shelly, Cancedda et al. 2007). The role of LKB1-STRAD in dendrite development in embryonic cortical neurons, is unknown. Interestingly, in *C. elegans* motor neurons, the nematode LKB1 counterpart Par-4, regulates dendrite growth downstream of UNC6/netrin signaling (Teichmann and Shen 2011). In cortical and CA1 hippocampal neurons, the LKB1/STRAD/STK25/GM130 complex might initiate axon development through regulation of Golgi localization, while the Reelin/Dab1 signaling might antagonize these events in favor of dendrite development (Matsuki, Matthews et al. 2010). Furthermore, in the embryonic cortex, defects in axon and dendrite polarity following LKB1 deletion were accompanied by alteration in centrosome positioning (Asada, Sanada et al. 2007). Through regulation of spatial localization of critical cellular organelles, centrosome and Golgi, and subsequent effects on cytoskeleton rearrangement and polarity, and trafficking of membrane components and cytoplasmic proteins, the STK25-STRAD-LKB1 complex might regulate fundamental aspects of cell-polarity that determine neurite formation and remodeling. These events might ultimately lead to unique patterns of axon and dendrite development in specific neuronal types. It is possible that in DGCs, axon specification occurs because of Golgi localization to, and specification of the primary dendrite, allowing retention of a single axon and elimination of all other neurites. These questions remain for future investigation.

While exposure of cultured neurons to Kainic acid or hyperexcitable conditions might induce Golgi fragmentation, the direct effect of Golgi manipulations in DGCs on promoting seizure activity in the hippocampus, remains to be determined. Alternatively, aberrant hilar and GCL neurite persistence and recurrent connectivity in the hilus and within the GCL upon manipulations of the STK25-Golgi complex, might promote recurrent excitation of these neurons. The abnormal hilar migration of a subpopulation of these neurons, could further augment seizure activity. In support, adult-born epileptic DGCs display abnormal migration into the hilus and severe defects in dendrite morphology (Parent, Yu et al. 1997, Overstreet-Wadiche, Bromberg et al. 2006). The cause and effect relationship between the STK25 complex, Golgi fidelity, and epilepsy, remain for future investigation.

**Note:** there is a typo in the second paragraph, page 2. The sentence that reads:
*"Furthermore, STK25 was found to colocalize with the Golgi marker GRASP62 (Rao et al., 2018, their Fig. 5C,D)."*
The Golgi marker is GRASP65, not GRASP62

***References***


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