ABSTRACT

Kinesins are molecular motors involved in intracellular transport directed towards the plus-end of the microtubules, using energy derived from ATP hydrolysis. Kinesin-3 is one of the biggest kinesin superfamilies. Its members (KIF1, KIF13A, KIF13B, and KIF16B, except KIF14 and KIF28) are known to be superprocessive and are associated with important cellular functions like vesicle transport, endocytic pathways, cell division, signaling, and morphogenesis. Any defect in kinesin-3 transport can cause various neurodegenerative disorders and cancer (Soppina et al., 2014). One of the widely studied members of the KIF13 subfamily, KIF13A, is found to be involved with tubular recycling endosomes (REs) and helps in cargo recycling in association with Rab GTPases (Delevoye et al., 2014; Etoh and Fukuda, 2019). Another member of the same subfamily, KIF13B, is known to transport DLG and PIP-3 containing vesicles (Hirokawa et al., 2009). However, its other physiological functions are largely underexplored. In the present work, we show that akin to KIF13A, KIF13B (GAKIN) also form tubular recycling endosomes when expressed in HeLa cells. Interestingly, the number of RE tubules is significantly higher than KIF13A. Surprisingly, the co-expression of KIF13A and KIF13B showed significant co-localization on REs, although the molecular mechanism and significance of both the motors on the same cargo during RE biogenesis and recycling are largely unknown. Similar to KIF13A, KIF13B is also found to co-localize with Rab10 and Rab22A, which are known markers for recycling endosomes. Further, we did co-localization studies of KIF13B with other Rabs to study their role in its regulation. Also, we checked the interaction of these Rabs with domain fragments of KIF13B via pull-down assays.