We have previously shown that Flotillin-1, a protein associated with membrane lipid rafts, is required for cell proliferation. Here we show that it is critical for the maintenance of Aurora kinase B protein levels. Knockdown of Flotillin-1 induced aberrant mitotic events similar to those produced by Aurora B knockdown, causing a significant decline in Aurora B levels and the dephosphorylation of its substrates histone H3 and 4EB-P1. Flotillin-1 interacted with Aurora B through its SPFH domain in a complex distinct from the chromosome passenger protein complex, and the two proteins co-purified in nuclear, non-raft fractions. Finally, the depletion of Aurora B caused by Flotillin-1 knockdown was prevented by proteasome inhibitors, suggesting that Flotillin-1 is required to prevent the proteasome-mediated degradation of Aurora B. These observations are the first evidence for a function of Flotillin-1 outside of lipid rafts, suggesting that it plays a critical role in the maintenance of the pool of stable and active Aurora B that is available for its nuclear activity in mitosis, and its non-nuclear activity as a regulator of mTOR signaling.