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AIP1 functions as Arf6-GAP to negatively regulate TLR4 signaling.

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It has come to our attention that the assay conditions used for the GAP assays were inappropriate based on published work, and the *in vitro* results that AIP1 is an Arf6-GAP are questionable (Fig. 4*g*). Specifically, it is known that full-length Arf is purified with GDP tightly bound. Because Arf-GTP is the substrate for GAP, GDP must be exchanged for GTP to measure activity. Arf-GTP is unstable without a hydrophobic surface such as liposomes or detergent micelles; little full-length Arf-GTP accumulates without liposomes or detergent. (The kit that was used does not include detergent.) Hydrophobic surfaces also facilitate exchange. Even with liposomes or detergent, Arf and truncation mutants of Arf exchange nucleotide slowly. In the presence of 0.1–1 mM $MgCl_2$ (the condition recommended in the kit), little (<5%) or no exchange should occur over 60 min. Therefore, we would like to withdraw the *in vitro* GAP activity assay presented in Fig. 4*g*. However, all the *in vivo* results indicating that AIP1 regulates PIP_2 production and TLR4 signaling remain valid. We are sorry if the reported results of AIP1 in Arf6-GAP activity have misled other researchers.