Supporting Information

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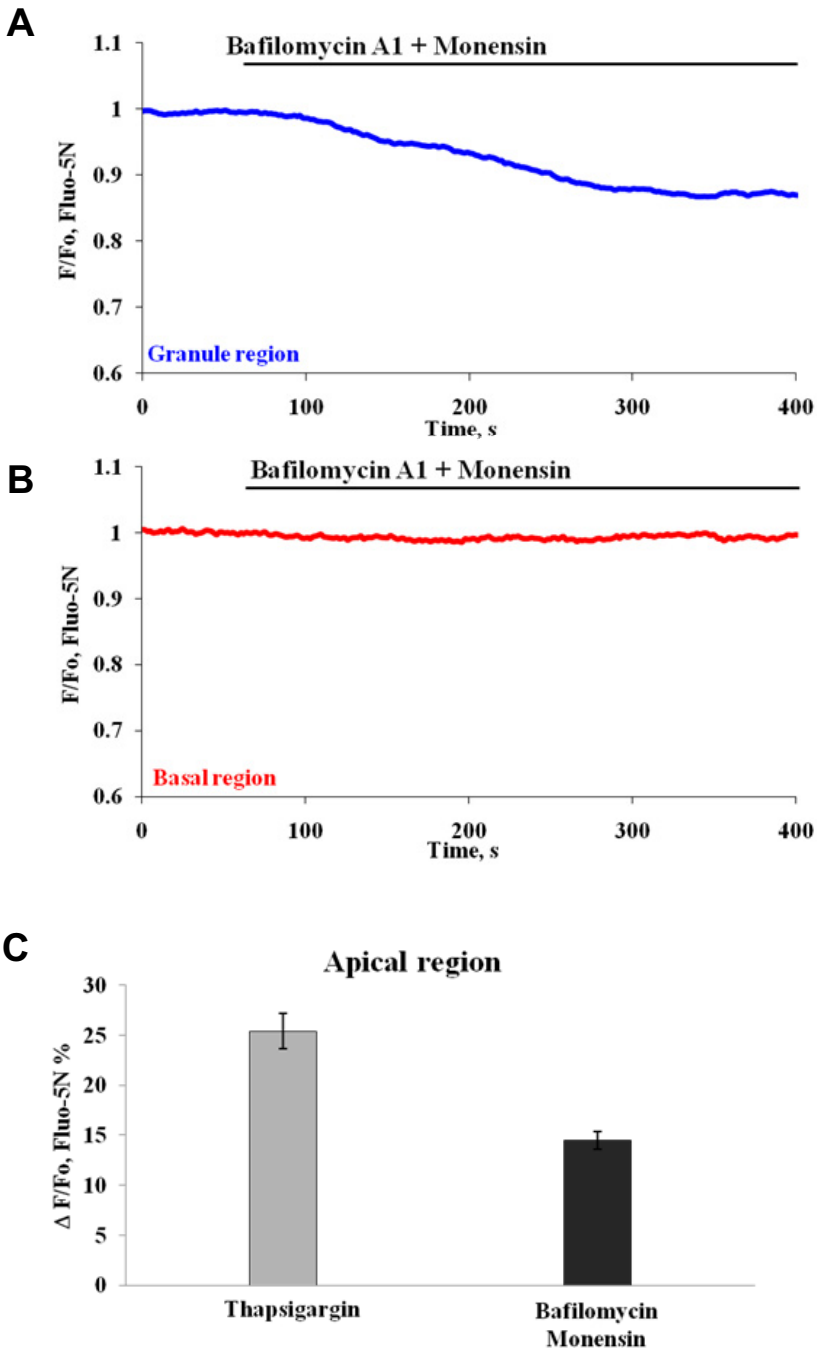

Fig. S1. Acid Ca^{2+} stores are exclusively present in the apical granular area. Application of bafilomycin A1 (100 nM) and monensin (5 μM) reduced $[Ca^{2+}]_{store}$ in the apical granular region (*A*), but not in the basal region (*B*) of the same cell; (*C*) compares the amplitudes of the reductions in $[Ca^{2+}]_{store}$ in the apical granular region evoked by thapsigargin and the bafilomycin/monensin mixture.

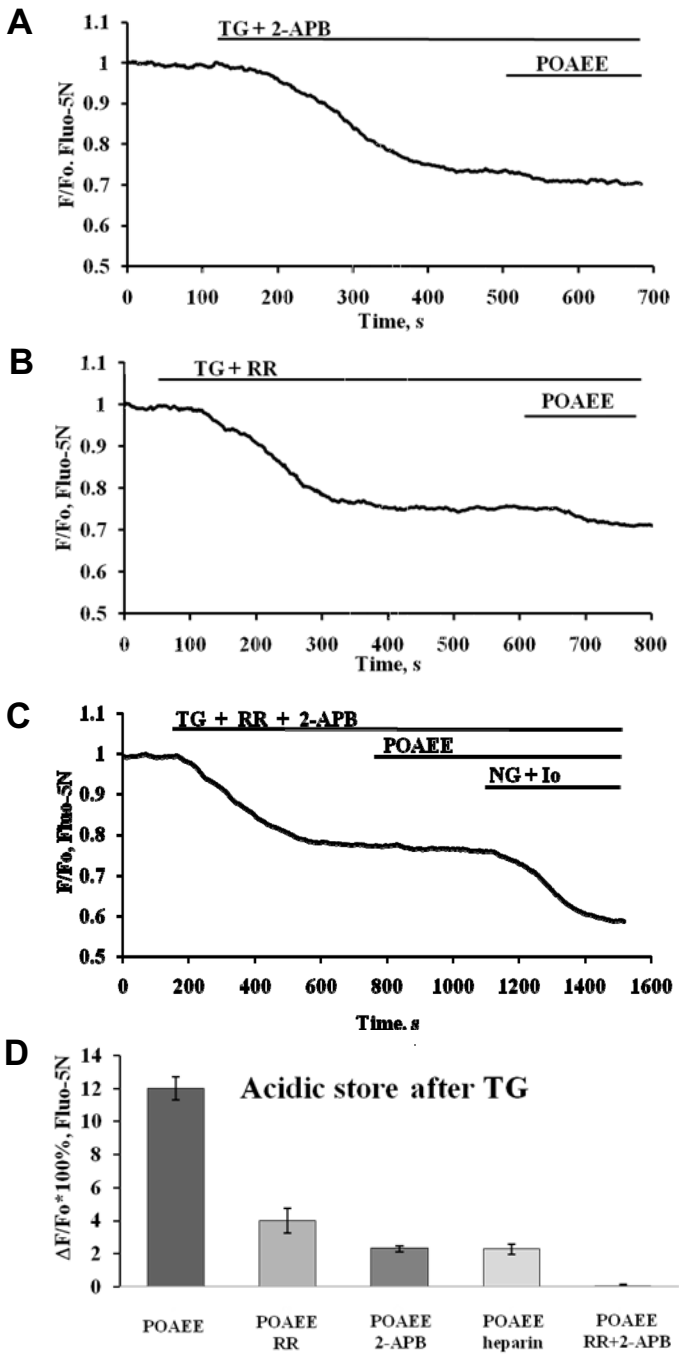

Fig. S2. IP_3Rs and RyRs are responsible for POAEE- induced Ca^{2+} release from the acidic stores. (*A*) When the IP_3R inhibitor 2-APB (100 μM) was applied together with thapsigargin (10 μM), the subsequent POAEE-induced Ca^{2+} release was very small [for quantification and comparison with results obtained without IP_3R inhibitor, see (*D*)]. (*B*) When the RyR inhibitor ruthenium red (RR, 10 μM) was applied together with thapsigargin (10 μM), the subsequent POAEE-induced Ca^{2+} release was small [for quantification and comparison with results obtained without RyR inhibitor, see (*D*)]. (*C*) When both the IP_3R (2-APB, 100 μM) and the RyR (RR, 10 μM) inhibitors were applied together with thapsigargin (10 μM), POAEE subsequently failed to elicit any further Ca^{2+} release [for quantification and comparison with the results obtained without IP_3R and RyR inhibitors, see (*D*)]. However, subsequent addition of nigericin (NG, 10 μM) and ionomycin (Io, 5 μM) elicited a substantial further reduction of [Ca^{2+}] in the acidic store. (*D*) Summary and quantification of the results obtained after TG treatment. Both RR and 2-APB substantially inhibited Ca^{2+} release from the acidic stores. When added together they abolished the POAEE-induced Ca^{2+} release.

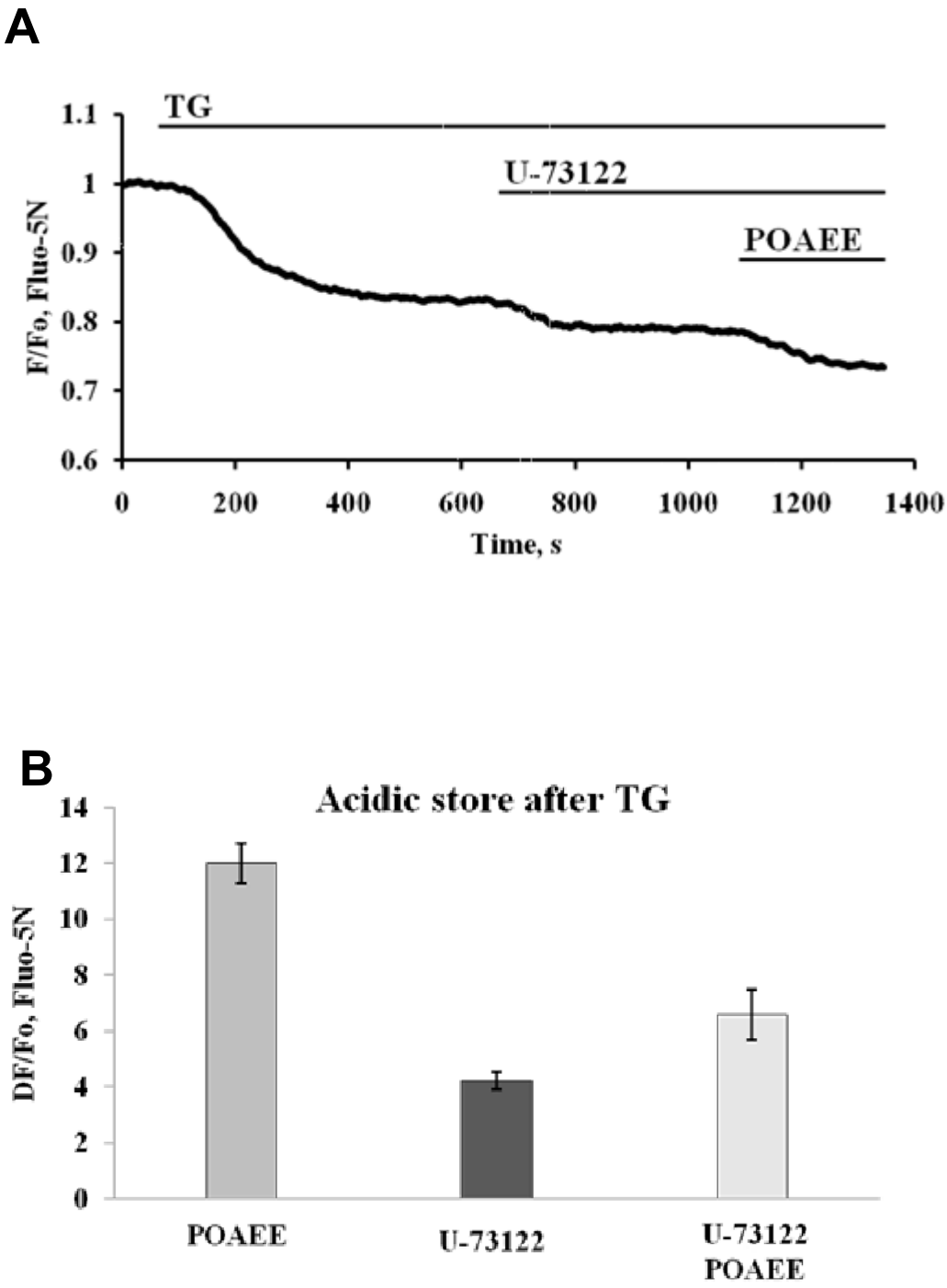

Fig. S3. The PLC inhibitor U73122 (10 μM) does not abolish POAEE-elicited Ca^{2+} release from the acid stores, but releases Ca^{2+} itself and then reduces the subsequent POAEE-induced Ca^{2+} release. (*A*) shows a typical trace. Thapsigargin induces the usual reduction in $[Ca^{2+}]_{store}$. After a plateau has been reached, U73122 causes a further reduction in $[Ca^{2+}]_{store}$, but thereafter POAEE (100 μM) is still capable of eliciting further Ca^{2+} release. (*B*) summarizes the data obtained.

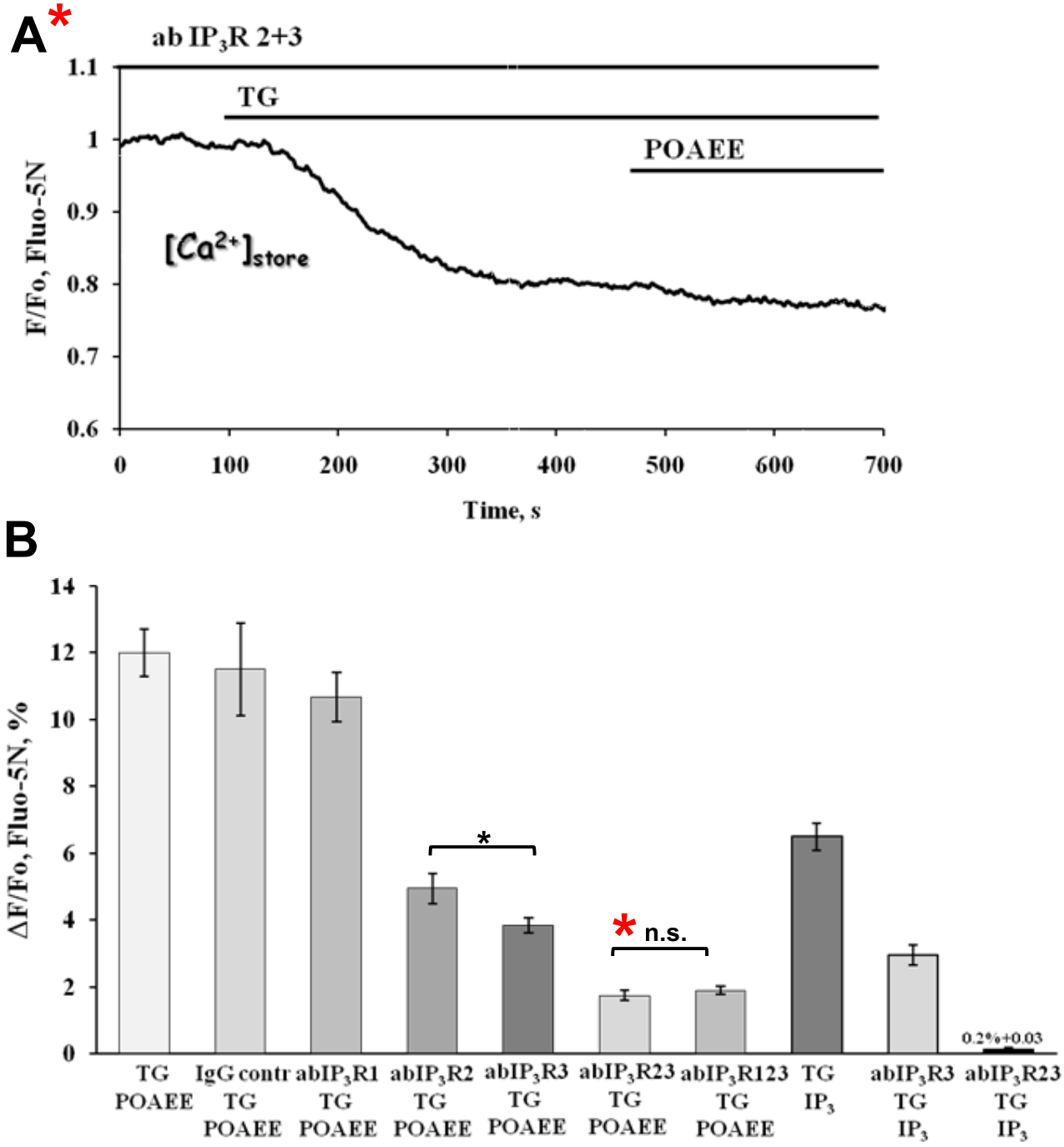

Fig. S4. Ca^{2+} release from acid stores induced by POAEE or IP_3 depend mainly on type 2 and 3 IP_3Rs. (*A*) POAEE only elicited a tiny reduction in $[Ca^{2+}]_{acid\ store}$ in presence of antibodies against type 2 and 3 IP_3Rs. (*B*) Summary of results concerning effects of antibodies against IP_3Rs of subtypes 1, 2, and 3, alone or in combinations, on POAEE- and IP_3-elicited reductions in $[Ca^{2+}]$ in the acid store (mean + SEM, $n = 4–6$ in each case).

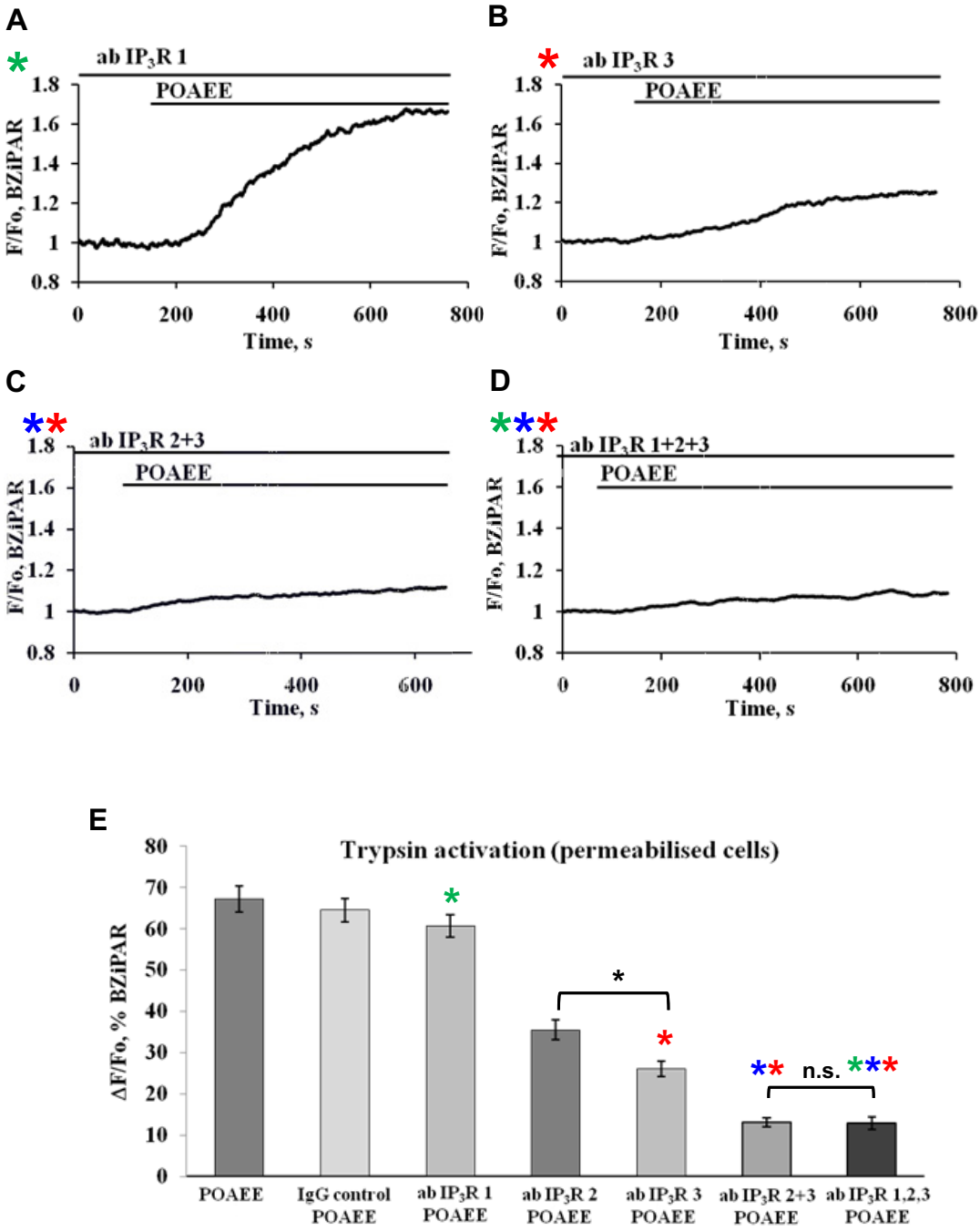

Fig. S5. POAEE-elicited trypsin activation depends on Ca^{2+} release from acidic stores through type 2 and 3 IP_3Rs. (*A*) Normal POAEE-elicited trypsin activation in presence of antibodies against type 1 IP_3Rs [similar to control, (*E*)]. (*B*) POAEE-elicited trypsin activation in presence of antibodies against type 3 IP_3Rs is markedly reduced compared to control (see *E*). (*C*) POAEE only evoked severely reduced trypsin activation in presence of antibodies against type 2 and 3 IP_3Rs [for quantification and comparison to control, see (*E*)]. (*D*) Antibodies against types 1, 2, and 3 IP_3Rs had same effect as antibodies to types 2 and 3 [see (*C*) and (*E*) for comparisons]. (*E*) Summary of data concerning effects of various antibodies against types 1, 2, or 3 IP_3Rs, alone or in combination, together with controls, on POAEE-elicited trypsin activation (mean ± SEM, $n = 5–6$ in each case).