Supplementary Table 1. Inhibitory properties of Pγ truncation mutants

Pγ mutants	cGMP as substrate		cAMP as substrate	
	IC_{50} (pM)	extent of inhibition (% of max.)	IC_{50} (pM)	extent of inhibition (% of max.)
Pγ1-70	14.6 ±28.1	37.3 ± 15.2	n.a	<10
Pγ1-73	39.2 ± 9.7	56.6 ± 3.7	28.6 ± 10.5	33.5 ± 3.3
Pγ1-76	21.8 ± 8.6	79.7 ± 7.5	45.1 ± 20.6	33.1 ± 3.9
Pγ1-80	25.3 ± 4.9	84.2 ± 4.5	26.3 ± 13.8	34.1 ± 4.7
Pγ1-83	38.0 ± 7.7	84.4 ± 5.0	21.8 ± 3.5	34.4 ± 2.4
Pγ1-84	31.7 ± 4.6	93.1 ± 4.8	29.1 ± 2.6	55.7 ± 2.0
Pγ1-85	32.7 ± 6.7	95.3 ± 7.2	21.2 ± 2.2	70.7 ± 3.0
Pγ1-86	20.9 ± 2.0	93.3 ± 4.3	21.0 ± 1.2	89.6 ± 2.6

The ability of a variety of Pγ truncation mutants to inhibit cGMP (1 μM) or cAMP (100 μM) hydrolysis was tested exactly as described in the legend to Fig. 4. Additional mutants not reported in Fig. 4 are included here as well. The IC_{50} was calculated from the dose-response curves by fitting the data to a 4-parameter logistic equation using Sigmaplot. The extent of inhibition was defined as the percentage of the original Pαβ activity inhibited by the mutant. Data represent the mean and S.D. of at least three separate experiments.

Note that the extent of inhibition with cGMP as a substrate is systematically higher in this study than for those Pγ mutants reported previously by Skiba et al. (1995). This difference is due to the different cGMP substrate concentrations used by Skiba et al. (0.5 mM) compared to the current study (1 μM). To confirm this, we re-tested several different Pγ mutants with millimolar levels of cGMP and observed maximal extents of inhibition [Pγ1-70; 20% inhibition; Pγ1-76, 45% inhibition; Pγ1-80, 60% inhibition] similar to those reported in Table III of Skiba et al. (1995).