Supporting Information

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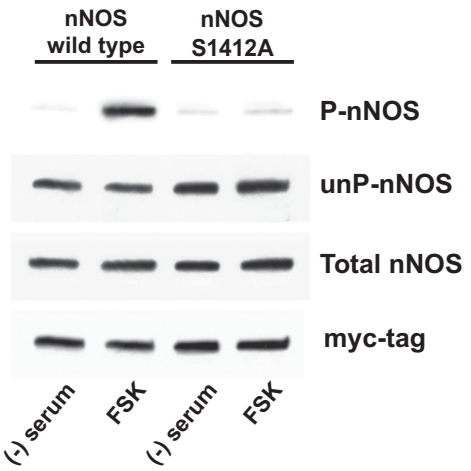

Fig. S1. Peptide antisera to phosphorylated and unphosphorylated neuronal NO synthase (nNOS) serine 1412 are sensitive and highly selective. LnCaP cells were transfected for 24 h with wild-type or S1412A myc-tagged nNOS, serum-starved for 24 h, then stimulated with forskolin (FSK) for 10 min before lysis. Unphospho-S1412-nNOS (UnP-nNOS) antibody shows a slight decrease for preparations with increased phospho-S1412-nNOS (P-nNOS), but total nNOS (commercial anti–N-terminus nNOS antibody) and anti-myc antibodies show even expression.

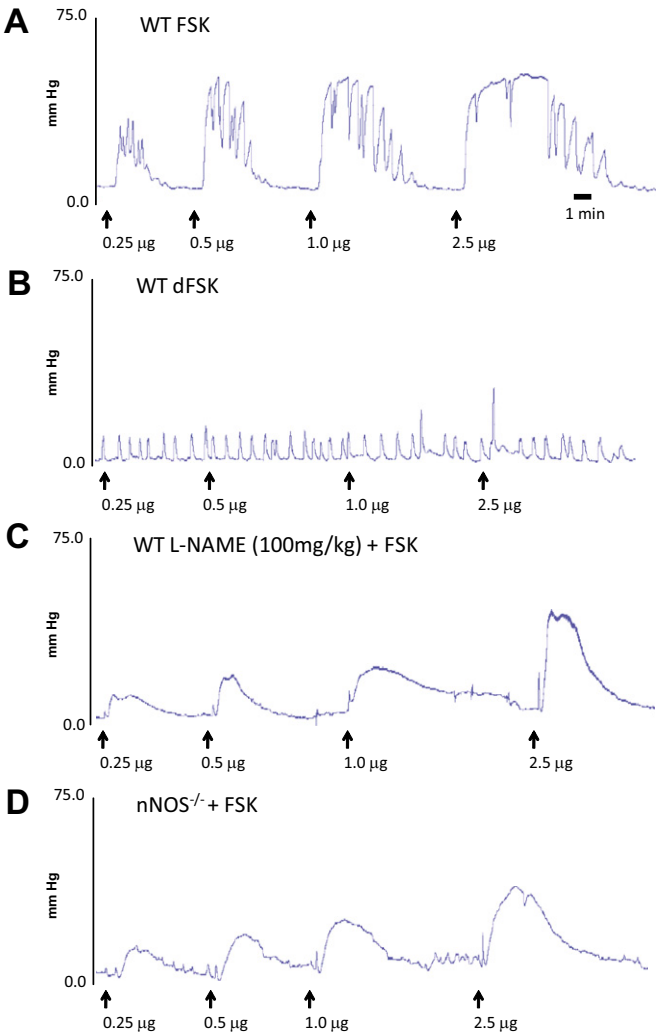

Fig. S2. (*A–D*) Representative tracings show overall dose-response for intracavernosal injection of FSK or deoxy-FSK (dFSK) in wild-type and nNOS$\alpha^{-/-}$ mice. Increased intracavernosal pressure (ICP) is recorded after injecting the indicated amount of FSK or dFSK in wild-type or nNOS$\alpha^{-/-}$ mice, or in WT mice pretreated with 100 mg/kg L-nitro-arginine-methylester (L-NAME). Responses to FSK injection are similar in L-NAME–treated wild-type and nNOS$\alpha^{-/-}$ animals.

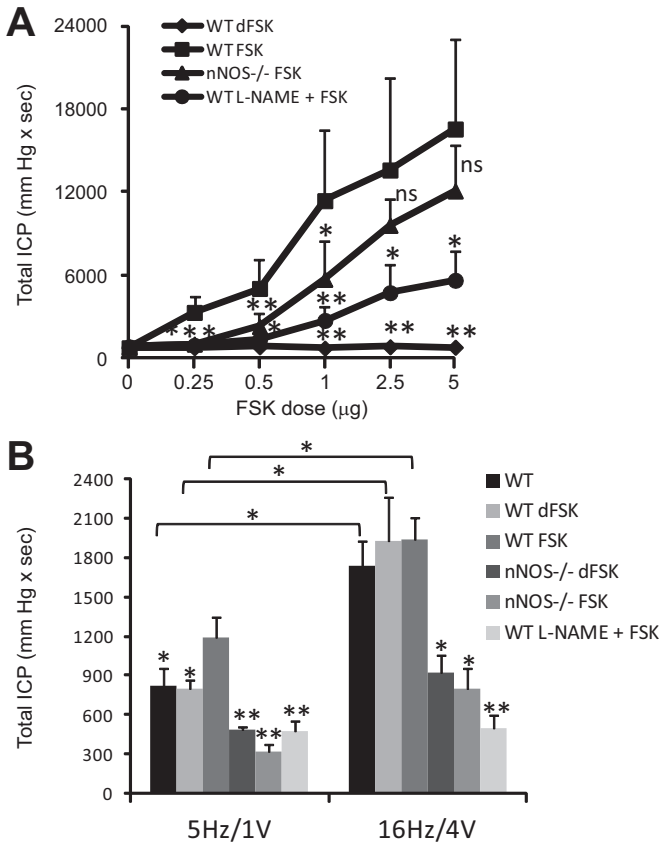

Fig. S3. (*A* and *B*) Total area under the curve (AUC; total ICP) for the experiments presented in Fig. 5. AUC analysis gives similar results as for maximal ICP. At low doses, intracavernosal FSK effects are inhibited by L-NAME and by nNOSα deletion. FSK also increases the response to submaximal cavernous nerve stimulation, but that effect is absent in nNOSα$^{-/-}$ mice and in wild-type mice pretreated with L-NAME. Because the effect of the general NOS inhibitor, L-NAME, is the same as the effect in nNOSα$^{-/-}$ mice, the FSK changes in ICP are likely mediated by nNOS rather than eNOS. Data are mean ± SE for $n = 6–9$ animals. *$P <$ 0.05 vs. wild-type FSK; **$P < 0.001$ vs. wild-type FSK. For 5 Hz vs. 16 Hz comparisons, *$P < 0.05$ by Student's t test. ns, not significant.