MOL PHARM

Correction to "Enhancement of long-term potentiation by a potent nitric oxide-guanylyl cyclase activator, 3-(5-hydroxymethyl-2-furyl)-1-benzyl-indazole"

In the above article [Chien W-L, Liang K-C, Teng C-M, Kuo S-C, Lee F-Y, and Fu W-M(2003) *Mol Pharmacol* **63:**1322–1328], Figs. 3 and 4 were incorrect due to a printing error. The correct versions of both figures appear below.

We regret this error and apologize for any confusion or inconvenience it may have caused.

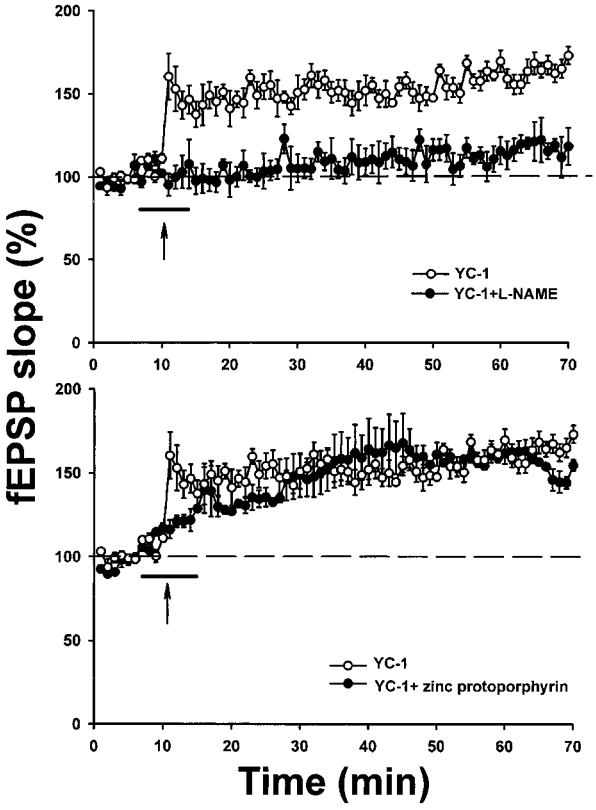

Fig. 3. NO-dependent enhancement of LTP by YC-1. LTP induced by one train of 50 Hz for 0.5-s tetanization (arrow) in the presence of YC-1 in hippocampal slice was inhibited by concomitant perfusion of NO synthase inhibitor L-NAME (300 μM; a) but not by the heme oxygenase inhibitor zinc protoporphyrin (1 μM; b). Horizontal bar represents the perfusion period of drugs.

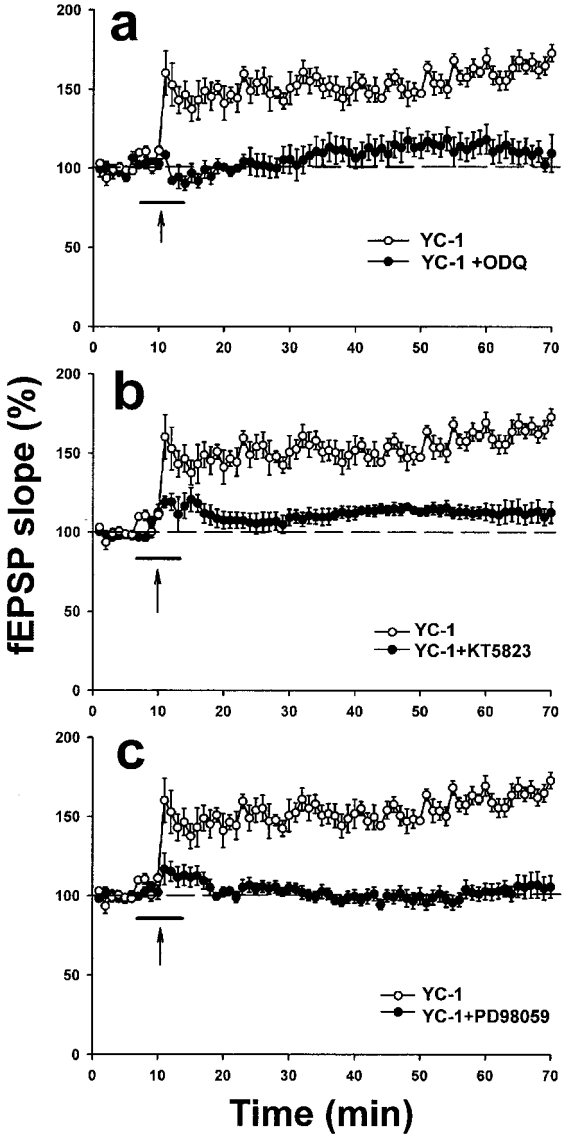

Fig. 4. Guanylyl cylase, PKG, and ERK are involved in the potentiation of LTP by YC-1. LTP induced by one train of 50 Hz for 0.5-s tetanization (arrow) in the presence of YC-1 in hippocampal slice was inhibited by concomitant perfusion of the guanylyl cyclase inhibitor ODQ (5 μM; a), PKG inhibitor KT5823 (2 μM; b), or ERK kinase inhibitor PD98059 (10 μM; c). Horizontal bar represents the perfusion period of drugs (n = 57).