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• 計畫英文名稱	Inhibition of Carbachol-stimulated Phosphoinositide Breakdown by Excitatory Amino Acids
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• 中文關鍵字	興奮性胺基酸；胺甲醯膽鹼；磷酸肌酐；麩胺酸鹽；鈉離子
• 英文關鍵字	Excitatory amino acid；Carbachol；Phosphoinositide；Glutamate；Sodium ion
• 中文摘要	查無中文摘要
• 英文摘要	Quisqualate, an excitatory amino acid receptor agonist, elicited biphasic effects on phosphoinositide (PI) breakdown in rat brain cortical slices. Veratridine, an Na/sup +/ channel activator, also evoked a similar biphasic effect. Quisqualate (0.1-1.mu.M) stimulated PI breakdown in a dose dependent manner. The maximal response of this effect was at 1.mu.M and the EC/sub 50/ was 0.3.mu.M. Veratridine-stimulated PI breakdown was maximal at 3.mu.M and the EC/sub 50/ was 1.mu.M. The quisqualate-stimulated IP/sub 1/accumulation was not affected by removal of extra cellular calcium whereas veratridine-stimulated effect was calcium dependent. Increasing both veratridine and quisqualate concentrations inhibited the stimulatory effects. The inhibition seen at higher quisqualate concentrations (1-10.mu.M) appeared to be due to activation of ionotropic glutamate receptors because the inhibitory effect was mimicked by kainate and .alpha.-amino-3-hydroxy-5-methyl-4-isoxazolepropionate (AMPA) and was abolished by the addition of ionotropic receptor antagonists, CNQX and MK-801. Veratridine inhibited its stimulatory effect at higher concentrations (3-100.mu.M), the IC/sub 50/ of this effect was about 30.mu.M. The inhibition was abolished by the presence of tetradotoxin, an Na/sup +/ channel blocker, suggesting Na/sup +/influx per se may inhibit agonist-stimulated phosphoinositide breakdown. Veratridine also inhibited quisqualate-stimulated IP/sub 1/ accumulation. The inhibitory effect has an IC/sub 50/ of 0.1.mu.M and was overridden by the stimulatory effect seen at 3.mu.M veratridine. These data suggested that higher concentrations of quisqualate cross react with

the ionotropic glutamate receptors and inhibited the metabotropic effect possibly through a mechanism linked to Na/sup +/ influx.