

**MECHANISMS OF [³H]GLYCINE RELEASE FROM MOUSE SPINAL CORD
SYNAPTOSOMES SELECTIVELY LABELLED THROUGH GLYT2
TRANSPORTERS**

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The release of glycine has been the object of very few studies. The aim of the present work was to characterize the release of the amino acid from spinal cord glycinergic nerve endings selectively prelabelled through glycine transporters of the GLYT2 type. Purified mouse spinal cord synaptosomes were incubated with [³H]glycine in the presence of the GLYT1 blocker NFPS and exposed in superfusion to varying concentrations of KCl, 4-aminopyridine (4-AP) or veratridine. KCl (≤ 15 mM), 4-AP (up to 1 mM) and veratridine (≤ 0.3 μ M) provoked [³H]glycine release by external Ca^{2+} -dependent, BoNT/C₁-sensitive, exocytosis. The overflows evoked by higher concentrations of K⁺ or veratridine involved external Ca^{2+} -independent mechanisms of different nature. Only the overflow evoked by 3 or 10 μ M veratridine occurred totally (3 μ M) or in part (10 μ M) by transporter reversal, being sensitive to the GLYT2 blockers Org 25543 or ALX 1393; in contrast, the external Ca^{2+} -independent [³H]glycine overflow provoked by 50 mM K⁺ was transporter-independent. This component of K⁺-evoked overflow, as well as the GLYT2-independent portion of the 10 μ M veratridine-evoked overflow, were largely sensitive to the vesicle depletor bafilomycin and completely prevented by blocking the mitochondrial Na⁺/Ca²⁺ exchanger with CGP 37157, indicating the involvement of exocytosis triggered by intraterminal mitochondrial Ca^{2+} ions.