



Overexpression of complexin in PC12 cells inhibits exocytosis by preventing SNARE complex recycling

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Abstract

Complexin is an important protein that functions during Ca^{2+} -dependent neurotransmitter release. Substantial evidence supports that complexin performs its role through rapid interaction with SNARE complex with high affinity. However, α -SNAP/NSF, which can disassemble the *cis*-SNARE complex in the presence of MgATP, competes with complexin to bind to SNARE complex. In addition, injection of α -SNAP into chromaffin cells enhances the size of the readily releasable pool, and mutation disrupting the ATPase activity of NSF results in the accumulation of SNARE complex. Thus, whether high concentrations of complexin could result in a reverse result is unclear. In this paper, we demonstrate that when stably overexpressed in PC12 cells, high levels of complexin result in the accumulation of SNARE complex. This in turn leads to a reduction in the size of the readily releasable pool of large dense core vesicles. These results suggest that high levels of complexin seem to prevent SNARE complex recycling, presumably by displacing NSF and α -SNAP from SNARE complex.

Key words

complexin SNARE complex cycle readily releasable pool
neurotransmitter release

Abbreviations

LDCV

large dense core vesicle

NSF

N-ethylmaleimide-sensitive fusion protein

RRP

readily releasable pool

α -SNAP

α -soluble N-ethylmaleimide-sensitive factor attachment protein

SNAP-25

synaptosomal-associated protein of 25 kD

SNAREs

soluble N-ethylmaleimide-sensitive fusion protein attachment protein receptors

VAMP

vesicle associated membrane protein, or synaptobrevin

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