Calcium channel subtypes differentially regulate fusion pore stability and expansion

Ardiles, Alvaro O.

González-Jamett, Arlek M.

Maripillán, Jaime

Naranjo, David

Caviedes, Pablo

Cárdenas, Ana María

Various studies have focused in the relative contribution of different voltage-activated Ca2+ channels (VACC) to total transmitter release. However, how Ca2+ entry through a given VACC subtype defines the pattern of individual exocytotic events remains unknown. To address this question, we have used amperometry in bovine chromaffin cells. L, N, and P/Q channels were individually or jointly blocked with furnidipine, ?-conotoxin GVIA, ?-agatoxin IVA, or ?-conotoxin MVIIC. The three channel types contributed similarly to cytosolic Ca2+ signals induced by 70 mmol/L K+. However, they exhibited different contributions to the frequency of exocytotic events and they were shown to differently regulate the final steps of the exocytosis. When compared with the other VACC subtypes, Ca2+ entry through P/Q channels effectively induced exocytosis, it decreased fusion pore stability and accelerated its expansion. Conversely, Ca2+ entry through N channels was less efficient in inducing exocytotic event