

Decapitation ischemia-induced release of free fatty acids in mouse brain. Relationship with diacylglycerols and lysophospholipids

林賜恩

Sun GY;Lu FL;Lin SE;Ko MR;

摘要

Abstract

In this study, the release of lysophospholipids (to depict phospholipase A2 activity) and diacylglycerols (DG) (to depict stimulated hydrolysis of polyphosphoinositides) was related to the decapitation-induced release of free fatty acid (FFA) in the mouse brain. To assay for lysophospholipids, Balb/c mice were injected intracerebrally with either [3H]choline or [3H]inositol for 16 h in order to label their respective phospholipids. These lipids were examined at various times (30 s to 30.5 min) after decapitation. Between 30 s and 1.5 min after decapitation, the rate of FFA release (3 micrograms FA/mg FA in phospholipids/min) was three times more rapid than that between 10 and 15 min (0.8 microgram FA/mg FA in phospholipids/min). FFA released during the initial phase were enriched in 20:4 and 18:0 whereas those released during the latter phase were nonspecific. The DG fatty acids are enriched in 18:0 and 20:4. Ischemia induced a rapid release of DG as measured by its fatty acid content (3.2 micrograms FA/mg FA in phospholipids/min). Unlike FFA, the level of DG reached a plateau after 1.5 min and remained elevated for the entire 30.5 min. In agreement with previous notions indicating the involvement of phospholipase A2 in ischemic insult, steady increases in radioactivity of both lysophosphatidylcholines and lysophosphatidylinositols were observed with time after decapitation. Based on the preferential increase in both 18:0 and 20:4 during the initial time period, the results suggest that poly-PI hydrolysis coupled to DG-lipase may contribute to the initial release of FFA, whereas the FFA released subsequent to the initial phase may be mainly a result of activation of phospholipase A2 acting on phosphatidylcholines and phosphatidylinositols.