**LIPID LYSOPHOSPHATIDYLCHOLINE-INDUCED Ca2+ INFLUX WAS MUCH MORE SUBSTANTIAL AND UNLIKELY STORE-OPERATED Ca2+ ENTRY (SOCE) IN CYTOTOXICITY OF MOUSE CEREBRAL MICROVASCULAR ENDOTHELIAL CELLS**

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**Objective:**Endothelial cell (EC) dysfunction and cell death have been observed in atherosclerosis and the lipid lysophosphatidylcholine (LPC) in oxidized low density lipoprotein has been implicated in such dysfunction and cell death. LPC reportedly interferes with Ca2+ signaling and NO production in EC and also caused EC apoptosis. Although there have been works on LPC toxicity in many EC types, there is hitherto no report on LPC-induced cytotoxicity in brain EC. In this work, we investigated the effects of LPC on mouse cerebral cortical endothelial cells (bEND.3 cells) as well as the influence of Ca2+ channel and cytosolic Ca2+ level.

**Method:**Brain microvascular bEND.3 cells cultured in Dulbecco’s modified Eagle’s medium (DMEM). Cytosolic Ca2+in bEND was measured with Fura-2 method. Mitochondria membrane potential (MMP) measured by MMP-Assay Kit. Cell viability was measured By MTT-assay. TUNEL assay for apoptotic cells. The p < 0.05 were considered significant (ANOVA). **Results:** LPC concentration-dependently caused cell death, the half-lethal concentration was approximately 50 μM . TUNEL assay shown LPC would cause cell apoptotic change. 50 μM LPC caused a substantial rise in cytosolic Ca2+ in Ca2+ -containing bath solution; the rise of Ca2+ was much smaller in Ca2+-free solution. This suggests LPC caused intracellular Ca2+ release and a large Ca2+influx. By contrast, the Ca2+ signals triggered by sarcoplasmic-endoplasmic reticulum in Ca2+-ATPase (SERCA) inhibitor cyclopiazonic acid (CPA) in Ca2+-free and Ca2+ -containing solution were much smaller. Since a maximal concentration of CPA was used (30 μM), the CPA-triggered Ca2+ influx was considered maximal store-operated Ca2+ entry. Obviously, LPC-induced Ca2+ influx was much more substantial and unlikely store-operated Ca2+ entry (SOCE).

**Conclusion:**All of the work on LPC cytotoxic actions on EC has focused on EC other than brain microvessel EC. Our results shown LPC caused Ca2+ release and Ca2+ Influx via unusual mechanisms in bEND.3 cells. To get much understanding the molecular mechanism of LPC cytotoxic actions on brain microvessel EC may have beneficial and helpful for the prevention or ameliorate disease treatment.