respectively, then embedded in para n-wax and cut into 5 m sections, nally xed on the glass slides coated poly-L-lysine.

PKC isoenzyme oligo-nucleotide probe sequences:

Objects	Probe Sequence
	5´--CAGGA CGTGG CCAAC CGCTT CGCCC GCAAA--3´
3 . & ù	5´--GAATG ACTTC ATGGG ATCCC TTTCC TTTGG--3´
	5´--CCAGC CTCTG CGGAA TGGAT CACAC TGAGA--3´
	5´--CCTTC AACTC CTATG AGCTG GGCTC CCTGC--3
3 . & û	5´--CGCGT GATCC AGATT GTGCT AATGC GGGCA--3´
	5´--TGGCT TCTCC TTTGT CAACC CCAAA TTCGA--3
	5´--GGAGA CATCC GCCAG CACCC TTTGT TTCGG--3´
3 . & ,	5´--GTGAA ATCAC CATTT GACTG CAGCA ATTTC--3´
	5´--CAGAG CACTG ATCAA CAGCA

values were signi cantly negative correlated to the absorbance values of PKC- , , and mRNA in situ hybridization, the correlation coe cient r were -0.695, -0.743 and -0.494, were <0.01, P<0.01 and P<0.05, respectively.

Morphological changes in lung tissue

Light microscope: In Sham group, the pulmonary interstitium and pulmonary alveoli are relatively complete and no in ltrated in ammatory cells. In PIR group, atelectasis coupled with emphysema, as well as pulmonary edema and widened, in ammatory cell in ltration were observed, and the pulmonary alveoli were lled with blood components e used, which indicated obvious injury. In PIR+PPF group, the lung injury and the in ammatory cells in ltrated were more slight and less than that of PIR group, and the structures of the alveoli were relatively intact.

Electron microscope: In PIR group, the endothelial cells with numerous pinocytosis vesicles were found in small pulmonary artery, and mitochondria became swelling and even vacuolar. Basilar membrane edema and vacuole degeneration, the dangling endothelial cells, and capillary lumen lled with PMed w3(a)19(p)1-5(l)3()-2oatt

ATP-sensitive potassium ion channels, and so on. e key researches [6] in the past have shown that PKC can induce broblast growth factor, reduce endothelial cells apoptosis resulted from the radiation injury. Tanigaki et al. [7] also found that the inhibitor of PKC, H-7 can induce acute lung injury. is study showed that PKC- , , and were negatively correlated to the indicators, the W/D and IQA, which were more sensitive to re ect the extent of damage for lung tissues, indicated that PKC isozyme had lung cells protection during ischemia-reperfusion injury [8]. e results of this study showed that W/D and IQA in PIR group signi cantly increased, the abnormal changes of the morphological structure were found. By contrast, W/D and IQA in PPF group only slightly increased and were signi cantly lower than that of PIR group, and abnormal morphological changes in lung tissues also markedly decreased. So the study indicated that propofol had an obvious protective role on ischemia-reperfusion injuries in lung cells. As shown in the gures and tables, PKC- , , and mRNA expression in PIR+PPF group were signi cantly higher than that of sham group and PIR group, MDA concentration was signi cantly lower than PIR group, SOD activity and NO levels were signi cantly higher than PIR group. Besides, the linear correlation between MDA, SOD, NO and PKC- , , and mRNA expression were signi cant, which indicated that propofol can cause PKC translocation and activation, protein substrates phosphorylation and enhance the activities of the endogenous antioxidant enzymes, iNOS and so on through certain signal transduction pathways, which alleviated ischemia reperfusion injuries by playing its cell protection and inducing the increase of the endogenous vasodilation mediators, such as NO. Kahraman et al. [9] also held the view that propofol can e ectively prevent PIRI by decreasing the level of oxygen free radicals, reducing lipid peroxidation. Meanwhile, propofol can directly react with free radicals to generate 2.6-isopropyl-p-groups and at the same time cause the inactivation of free radicals. Murphy et al. [10] believed that propofol mainly interfered with the process of hydrogen abstraction during lipid peroxidation to