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镉对河南华溪蟹肝胰腺线粒体的影响

Effects of cadmium on the hepatopancreatic mitochondria of the freshwater crab *Sinopotamon henanense*

关键词：河南华溪蟹 肝胰腺 线粒体 SDH 线粒体膜电位 镉

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摘要：运用透射电镜技术、电镜细胞化学技术、酶组织化学技术和JC-1荧光染色,观察不同浓度镉对河南华溪蟹(*Sinopotamon henanense*)肝胰腺线粒体的损伤.实验设对照组和5个染毒组:3.56、7.12、14.25、28.49和56.98 mg·L^{-1},处理时间为72 h.透射电镜观察显示,在低浓度组,线粒体轻度肿胀,部分线粒体外膜破裂,随染毒浓度的增加,线粒体水肿增加,嵴缩短甚至消失,直至线粒体破裂.酶组织化学检测显示,琥珀酸脱氢酶(succinic dehydrogenase,SDH)主要在细胞浆中表达,呈蓝紫色颗粒.SDH活性在3.56、7.12和14.25 mg·L^{-1}浓度组中比对照组显著增加($p < 0.05$),而在28.49和56.98 mg·L^{-1}浓度组中没有明显变化.SDH的亚细胞定位结果与酶组织化学检测的结果一致.JC-1荧光强度在3.56、7.12和14.25 mg·L^{-1}浓度组比对照组显著增加($p < 0.05$).线粒体膜电位与SDH的活性有正相关性(r=0.736, $p < 0.05$).结果显示,镉引起线粒体结构损伤、功能障碍,并与镉浓度具有剂量-效应关系.线粒体是镉细胞毒性的重要靶位.

Abstract: The present study aims to investigate the effect of cadmium on the mitochondria in the hepatopancreas of the freshwater crab *Sinopotamon henanense* by transmission electron microscopy, electron microscopic cytochemistry, enzymhistochemistry and JC-1 staining. Crabs were exposed to various Cd concentrations of 0 (control group), 3.56, 7.12, 14.25, 28.49 and 56.98 mg·L^{-1} for 72 h. The damages of mitochondria, activities of SDH and mitochondrial membrane potential were measured. There were variations in the mitochondria with swelling, membrane disruption, shortening or disappearance of cristae and mitochondrial rupture. The mitochondrial dropsy magnified and mitochondrial damages were facilitated with increased Cd concentration. Enzymhistochemistry revealed that SDH with blue granules were expressed in the cytoplasm. Activities of SDH increased significantly in the 3.56, 7.12 and 14.25 mg·L^{-1} Cd groups (compared to the control group, $p < 0.05$) but no significant changes occurred in the other two groups. The tendency of the SDH expression was similarly expressed in the electron microscopic cytochemistry compared to the enzymohistochemistry test. The fluorescence intensity of JC-1 increased significantly in the 3.56, 7.12 and 14.25 mg·L^{-1} Cd groups compared to the control group ($p < 0.05$). There was a positive correlation between the mitochondrial membrane potential and the SDH activities (r=0.736, p<0.05). The results indicated that Cd could induce mitochondrial damage and dysfunction in a dose-dependent manner. Therefore, mitochondria are proven to be a major cellular target of Cd toxicity.

Key words: *Sinopotamon henanense* hepatppancreas mitochondria SDH mitochondrial membrane potential cadmium

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