中文摘要

本論文主要研究目的在於發展淡水蜆(*Corbicula fluminea*)暴露於重金屬-銅污染之生物反應機制模式，以鰓膜介面為基礎探討鈉離子傳輸通量及傳輸膜電位與開闔行為反應之間的生理交互關係，做為預測水域環境之銅可獲取率之理論基礎。整合以生物配體模式(biotic ligand model, BLM)為基礎之銅可獲取率(bioavailability)、Michaelis-Menten 動力為基礎之鈉與銅離子傳輸通量及電化學為基礎之傳輸膜電位，發展淡水蜆電生理反應之機制模式，預測以鰓膜介面為基礎之受銅影響之生理毒性效應及閉殼反應。引用虹鱒(rainbow trout)暴露於銅污染之鈉離子傳輸抑制影響，及淡水蜆平衡/非平衡狀態下之鈉離子平衡/主動傳輸膜電位等相關文獻，驗證本論文提出之模式結果；模式預測結果與文獻實驗量測值極為接近，證明本研究機制模式預測之可靠度。評估之結果顯示，淡水蜆受到銅污染 0 至 0.2 μM 之生理毒性效應包含： (1) 鈉離子最大傳輸通量變化由 12.9 降至 0.4 μmol g^{-1} hr^{-1}，(2)體內鈉離子濃度變化由 10.5 降至 7.5 mM，同時(3)因鈉離子傳輸通量導致鰓膜二側之平衡及主動膜電位變化將分別由-84.2 去極化至 10 mV 及-8 去極化至 0 mV。本研究提出之模式架構進一步顯示： (1)造成抑制 50%鈉離子傳輸膜電位及傳輸通量之外部銅離子活性濃度分別為 0.072 與 0.043 μM，鍵結於鰓膜表面之銅離子與抑制生理反應之關係為 $3Cu^{2+}$:1 及 1，(2)預測淡水蜆最大銅離子吸收傳輸通量為 0.369 (95% CI: 0.26-0.51) μmol g^{-1} h^{-1}，半飽和親合常數為 7.87×10^{-3} (95% CI: 5.72×10^{-3}-11.20×10^{-3}) μM，及(3)呈現因地而異(site-specific)之應用性，預測在不同養殖地域之淡水蜆生理毒性效應。本論文提出之機制模式可提供一有效方法做為未來發展生物監測技術之基礎，進而提供水域重金屬污染之環境危害風險評估。

關鍵詞：淡水蜆；生物可獲取率；電生理；傳輸膜電位；鈉傳輸通量；銅；生態毒理

Abstract

The purpose of this thesis is to develop a biological response mechanistic model based on the gill-based physiological interactions among sodium (Na) transport, Na membrane potential and valve closure response in freshwater clam (*Corbicula fluminea*) in response to waterborne copper (Cu) for providing a tool to assess Cu-bioavailability. This study have integrated Cu bioavailability based on biotic ligand model (BLM), Na and Cu uptake flux based on Michaelis-Menten kinetics, and electrochemically-based gill potentials, to derive an electrophysiological response model of *C. fluminea* describing the action mechanisms of a gill-based membrane interface by which valve closure behavior and Cu toxicity can be predicted. To test the proposed model against published data regarding inhibition of Na uptake in rainbow trout in response to Cu and Na total/active transport in equilibrium/non-equilibrium conditions in *C. fluminea*. The predictions are reasonably agreed with published measured, confirming that the predictive model is robust. The results show the physiological toxic response including (i) inhibition of M-M maximum Na uptake rate is 12.9 to 0.4 μmol g^{-1} hr^{-1}, (ii) decrease of internal Na concentration is 10.5 to 7.5 mM, and (iii) depolarization of total and active transport are -84.2-10 and -8-0 mV, respectively in response to external Cu activity from 0 to 0.2 μM. This proposed framework captures the general features observed in the model applications including: (i) 50% inhibitory Cu^{2+} activities for Na membrane potential and uptake rate are estimated to be 0.072 and 0.043 μM, respectively, with a stoichiometry of $3Cu^{2+}$: 1 and 1 , (ii) predicted M-M maximum Cu uptake flux in C. fluminea is 0.369 (95% CI: 0.26-0.51) μmol g^{-1} h^{-1} with a half-saturation affinity constant of 7.87×10^{-3} (95% CI: 5.72×10^{-3}-11.20×10^{-3}) μM, and (iii) the site-specific clam gill potentials can be predicted in aquaculture settings. Here this study successfully provide a general

approach to harness the potential new biomonitoring techniques for assessing the environmental impact of waterborne Cu.

Keywords： *Corbicula fluminea* ; Bioavailability ; Electrophysiology ; Gill membrane potential ; Sodium transport ; Copper ; Ecotoxicology