Cardiotoxicity of Cinnabar in Zebrafish Embryos

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Cinnabar has been used as a sedative and soporific agent for more than 1,000 years in China, India and other Asian countries, nowadays, the safety of cinnabar is widely disputed for the presence of mercury sulphide (HgS). The underlying mechanism of action and potential embryonic toxicity remain unclear. The aim of this study was to determine the toxicity of cinnabar to selected organs and systems including motor neuron and heart and to arouse the public concern on using of cinnabar. Zebrafish embryos were used as to assess the toxicity risk by measuring mortality rate, locomotion behavior, phenotype of motor neuron and heart, and various heart function parameters. Embryos treated with 0-10 mg/ml for 6 days did not show lethal toxicity. There was no significant change of locomotion activity of the embryos and phenotype of motor neuron after treatment at 10 mg/ml; thus, behavior assay did not reflect the sedative effect. Surprisingly, pericardial edema was observed on the 3dpf embryos. Therefore, the cardiotoxicity was further assessed using a Tg:CMLC-GFP transgenic zebrafish which expressed green fluorescent protein in heart. Heart rate, stroke volume and cardiac output were decreased while fractional shortening was increased at 3-10 mg/ml. These results suggested that cinnabar exhibited cardiotoxicity in developing heart of embryos under non-lethal toxic concentrations. Without sedative effect of cinnabar on embryos, surprisingly, our results, for the first time, identified the previously unaddressed cardiotoxicity of cinnabar in the developing heart of zebrafish embryo. This result arouses safety concern on the use of this traditional medicine.