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## Itai-Itai Disease Caused by Cadmium Poison-

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Although the bone injury is secondary and treatable, renal injury is unrecoverable and deteriorates climaxing in end-stage renal failure in severe cases.

Although habitual Cd intoxication substantially results in a renal complaint, acute exposure to poisonous Cd boluses primarily results in liver damage. Acute Cd hepatotoxicity has been well studied in experimental creatures, and its medium has been illustrated in detail.8 Still, the liver of itai-itai complaint cases has not been the focus in exploration; thus, the goods of prolonged Cd exposure on the liver remain to be clarified. Our faculty, the University of Toyama, located in the region where an itai-itai complaint has passed, maintains a large collection of necropsy samples from itai-itai complaint cases. One of the points of the present study was to assess the histopathological characteristics of the liver of itai-itai complaint cases to estimate the goods of habitual Cd hepatotoxicity.

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## Commentary

Cadmium (Cd) is an environmental contaminant ranked as one of the most poisonous substances, and mortal artificial conditioning has markedly increased its distribution in the global terrain. Food is the major source of Cd exposure to the general population; thus, the goods of habitual exposure to this essence are a major concern for humans. Several studies reported that indeed habitual exposure to low Cd boluses can beget serious health goods. The mass cadmium poisoning in Toyama Prefecture, Japan, known as itai-itai sickness, began around 1912. Locals invented the phrase "Itai-Itai disease" to describe the intense pains people with the disorder experienced in their spines and joints. The most severe form of chronic Cd poisoning is an itai-itai illness, which is caused by persistent oral Cd consumption. It struck a large number of residents of the Jinzu River valley in Toyama Prefecture, Japan, an area heavily contaminated by Cd from a zinc mine upstream. Cadmium (Cd) is a hazardous heavy metal that is widely distributed in the environment and is extremely hepatotoxic. In experimental animals, acute Cd hepatotoxicity has been widely investigated; however, the consequences of sustained Cd dosages on the liver are unknown. We investigated materials from instances of itai-itai sickness, the most severe form of chronic Cd poisoning, to assess chronic Cd hepatotoxicity. To determine Cd content in organs, we compared 89 cases of itai-itai illness to 27 control cases. For histological examination, we looked at 80 instances of itai-itai illness and 70 control cases. We also used immunohistochemistry to look for metallothionein, a protein that binds to and detoxifies Cd. In the itai-itai illness group, hepatic Cd concentration was higher than Cd concentration in all other organs measured, whereas, in the control group, it was second highest after renal concentration. Fibrosis was found at a much greater rate in the liver of the itai-itai illness group than in the control group. Itai-Itai complaint is the most severe form of habitual Cd poisoning caused by prolonged oral Cd ingestion. It developed in multitudinous occupants of the Jinzu River receptacle in Toyama Prefecture, Japan, an area most oppressively defiled by Cd that began from a zinc mine located upstream. The main target organ of Cd toxin in the itai-itai complaint is the order, where the injury is manifested by tubular and glomerular dysfunction. Renal dysfunction causes insufficiency of active vitamin D, followed by bone injury conforming to a combination of osteomalacia and osteoporosis. Although femoral pain and lumbago are constantly seen as the original incarnation, the leakage of low molecular weight proteins is observed in urine tests from an earlier stage.