Abstract

Methyl mercury poisoning has been reported as Minamata disease in Japan. Visual dysfunctions have been an important aspect of human methyl mercury poisoning. In this study, fast axonal transport of S35 methionine in the retina, lateral geniculate nucleus, and superior colliculus of the visual system has been reported.

Sixty five male albino rabbits were divided into two groups. Methyl mercury (2.5 or 5.0 mg/kg body wt.) was injected to the experimental group for two weeks. Rabbit eyes were also injected S35 methionine. Rabbits were sacrificed after 2hr, 3hr, and 4hr. Optic pathway was dissected into segments, homogenized in SDS buffer. And radioactivity was counted.

Results have revealed that axonal transport of S35 methionine was accelerated and incorporation of S35 methionine in the proteins of lateral geniculate nucleus and superior nucleus was decreased. Most likely low levels of mercury in these regions resulted in the decreased biosynthesis of proteins. We may conclude that the perturbations of protein biosynthesis by methyl mercury poisoning, most likely, were responsible for the vision impairments.