SUSCEPTIBILITY OF VOLES IN HOKKAIDO AND THE INNATE RESISTANCE OF CLETHRIONOMYS RUFOCANUS BEDFORDIAE TO INFECTION BY THE CESTODE, TAENIA TAENIAEFORMIS

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Of the 377 specimens of Clethrionomys rufocanus bedfordiae, 43 Apodemus speciosus ainu and 72 Apodemus argenteus collected in the suburbs of Sapporo, Hokkaido, 2, 1 and 1 specimens were found to be infected with larval Taenia taeniaeformis in the liver, respectively. This fact confirmed that the vole can serve as an intermediate host of T. taeniaeformis. The infection rate, however, was very low. Experimental infection with T. taeniaeformis showed that A. argenteus was the most susceptible, followed by A. speciosus ainu and C. rufocanus bedfordiae. The susceptibility of A. argenteus to T. taeniaeformis was so high that the number and size of the liver cysts recovered were the same as those recovered from Wistar rat. A significantly smaller number and only a few cysts were recovered from A. speciosus ainu and C. rufocanus bedfordiae, respectively. Although experimental infection showed that C. rufocanus bedfordiae was resistant to T. taeniaeformis, the infection rate was observed to be significantly higher in C. rufocanus bedfordiae, which were caught in a specific area and then experimentally infected with T. taeniaeformis isolated from the same host.

The mechanism for the low susceptibility of C. rufocanus bedfordiae to T. taeniaeformis infection was investigated. Hatching rate of T. taeniaeformis eggs in the small intestine of C. rufocanus bedfordiae was compared with that of ICR mice. Although the hatching rate in C. rufocanus bedfordiae was lower than that in the mice and varied greatly among the individual hosts, it was observed that a sufficient number of eggs was hatched to give rise to viable infection. On days 1 to 3 post infection with T. taeniaeformis, postonchospheral larvae were observed in the C. rufocanus bedfordiae liver. However, on days 15 and 30 post infection the larva was no longer visually identifiable. Thus, it was suggested that in C. rufocanus bedfordiae, the T. taeniaeformis oncosphere was able to migrate to the liver but were destroyed there. Similar failure of the T. taeniaeformis oncosphere to develop in the liver of cortisone-treated C. rufocanus bedfordiae was also noted. From these observations, several reasons were suggested to explain the differences in the pathogenicity of T. taeniaeformis infection in ICR mice and Clethrionomys. 1. There were differences in the mechanisms of resistance to T. taeniaeformis infection in ICR mice and Clethrionomys. 2. In Clethrionomys, the hepatic larvae were destroyed much earlier than in ICR mice during the initial stage of the infection, thus resulting in different pathogenic lesions. 3. The administration of cortisone might not have immunosuppressed Clethrionomys.