A relatively high prevalence was consistently detected in the study period, indicating the establishment of urban cycle in the city. 2) A quarterly monitoring for the *E. multilocularis* prevalence among a local fox population was carried out in Koshimizu from April 1997 to January 1998. Thirty-six breeding dens of fox families were identified in the area and 534 fecal samples were collected within 500 m around the dens. Seasonally, whereas the prevalence of coproantigen positives showed no fluctuation (51.6-66.7%), the mean coproantigen OD values of positives rose in summer and winter. Similar variation was found in the egg positive rate in which higher prevalence was observed in summer and winter (31.3% and 38.7%, respectively) than spring and autumn (13.3% and 13.5%, respectively). The data implies the infection pressure on foxes increased in summer and winter.

Genetic and Developmental Study of Tattered^{hokkaido} (Td^{ho}) mouse

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Td^{ho} mouse is a newly found, X-linked dominant mutation which exhibits hyperkeratotic skin, reduced viability in affected females, dwarfism, growth retardation during weaning age, and prenatal lethality in affected males. To map this Td^{ho} locus, microsatellite markers of the proximal region on X chromosome were used. Linkage analysis suggested a possible gene order of cen- (Td^{ho}, *DXMit26, DXMit55, DXMit123*) *-DXMit161-DXMit54 -DXMit103 -DXMit52 -DXMit190 -DXMit138* on the X chromosome.

In the developmental study, it was found that the Td^{ho} male mutants died between E12.5 and E14.5. For examination of genes related to the cause of lethality in Td^{ho} male embryos, suppression subtractive hybridization and Northern blot analysis of globin mRNAs were performed at E12.5 embryos of Td^{ho} mutant and normal mice. Diminished expression levels of embryonic globin genes (ζ and ε globins) in the peripheral blood of E12.5 Td^{ho} male embryos were demonstrated. The increased apoptosis of yolk sac-derived erythrocytes was found at E12.5 Td^{ho} male embryos. Therefore, a defect of embryonic hematopoiesis was suggested to occur in Td^{ho} male embryos.

Ebp, which was identified as a responsible gene for *Td* mouse, was speculated as a candidate gene of Td^{ho}. Two point mutations were detected in the nucleotide sequence of *Ebp* of Td^{ho} mouse. These substitutions caused two amino acids exchanges in the third transmembrane domain of EBP. This mutation is considered to correlate with sterol content of plasma, cholest-8(9)en-3β-ol was accumulated in Td^{ho} female mutants. It was discussed how the toxicity of intermediate metabolites of cholesterol influenced em-

bryonic lethality and hematopoiesis in Td^{ho} males, and hyperkeratosis in Td^{ho} adult females.

In humans, it is also demonstrated that the mutations of EBP result in X linked dominant chondrodysplasia punctata (CDPX2). Therefore, Td^{ho} mutant mouse is expected to be one of useful animal model for CDPX2.

The original papers of this thesis appeared in "Mamm. Genome", Vol. 8, 578-580 (1997) and "Lab. Anim. Sci. ", Vol. 50, 8-12 (2000).

Molecular characterization and antiviral activity analysis of the interferon-inducible *Mx* gene in mice

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The mouse genome contains two related interferon-regulated genes, *Mx 1* and *Mx 2*. *Mx 1* codes for the nuclear 72-kDa protein that interferes with influenza virus replication after interferon treatment. On the other hand, the *Mx 2* gene is nonfunctional in all laboratory mouse strains examined, since its ORF is interrupted by insertional mutation and a subsequent frame shift. In the present study, the characterization and identification of IFN-inducible *Mx 1* and *Mx 2* genes in the feral-origin strains have been demonstrated.

On part 1, several mouse strains established from feral-origin mice were tested to determine their *Mx 1* + or *Mx 1* − allele status with PCR-RFLV, sequence analysis, RT-PCR and immunofluorescence staining. All of the mouse strains originating from feral-origin mice were found to uniformly carry the *Mx 1* + allele. Therefore, it is conceivable that the *Mx 1* + allele in feral-origin populations serves a function against some pathogens related to orthomyxoviruses. The PCR-RFLV and sequence analysis allowed to classify the *Mx 1* + alleles of the laboratory and feral-origin mouse strains into distinct classes. RT-PCR and immunofluorescence staining demonstrated that the *Mx 1* transcripts and proteins were induced by IFN α / β in macrophages from feral-origin mouse species.

On part 2, the author demonstrated that *Mx 2* mRNA of cells from the feral-origin mouse strains NJL (*Mus m. musculus*) and SPR (*Mus spretus*) differed from the nonfunctional mRNA of the laboratory mouse strains tested. The *Mx 2* mRNA of the feral-origin strains contained functionally a single long ORF consisting of 656 amino acids. The author further showed that Mx 2 protein in the feral-origin strains was expressed upon interferon treatment and localized to the cytoplasm much like the rat Mx 2 protein, which inhibited VSV replication. Furthermore, transfected 3 T 3 cell lines of laboratory mouse origin expressing *Mx 2* from the feral-origin strains acquired slight resistance to VSV.

In the study of part 3, the author has demonstrated that the embryonic fibroblastic