Abstract of Master's Dissertation

Course	MTM	Name	Kotaro Takahashi
Thesis Title	Mice deficient for GILT (gamma-interferon-inducible lysosomal thiol reductase) have resistance to the pathogenicity of cholera toxin.		

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Objective :

Our objective is to indicate that GILT (gamma-interferon-inducible lysosomal thiol reductase) enhances the virulence of cholera toxin.

This will provide a new model for the pathway by which cholera toxin is modified in cells. In addition, we aim to show that GILT plays a role otherwise the acquired immune system.

Method :

We intraperitoneally administered two times the 50% lethal dose of cholera toxin to GILT knockout mice and wild-type mice, and obtained mortality rate. The experiment was conducted on 12 wild-type mice and 13 GILT knockout mice. The mice were dissected and examined the toxicity macroscopically and pathologically.

Result :

All of the wild-type mice died, whereas 9 of the GILT knockout mice survived. Survival rate of the knockout mice was 70% (95%CI: 44-94%).

Macroscopically, wild-type mice showed collapsed intestine, whitening of the liver, and atrophy of the spleen. Surviving cases of GILT knockout mice showed none of these changes. However, in the dead cases of GILT knockout mice, similar changes were observed with the intestinal tract. As for the liver and spleen, there were not much change.

Histologically, no significant changes were observed in the intestinal tract and spleen. Wild-type mice showed the characteristic finding of necrosis of perivascular cells in the liver, but this finding was not observed in the surviving cases of GILT knockout mice. Abnormalities of hepatocytes were also observed in deceased GILT knockout mice, but the changes were not as pronounced as in wild-type mice.

＊ The abstract, containing the objective, method, result and conclusion should not exceed 300-500words and printed double sided on A4 paper)

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Conclusion :

GILT has been revealed to promote the pathogenicity of cholera toxin.

The results indicate that GILT acts in other ways than the mechanisms of antigen presentation in the acquired immune system. It is likely to be involved in protein degradation and inflammatory processes.

CT has toxic effects other than causing diarrhea in mice. Further studies are required to reveal how GILT acts in these effects.

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