INCIDENCES OF LYMPH–NODE SWELLING, PROTEINURIA AND SKIN LESION IN MRL MICE AND THEIR HYBRIDS

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Systemic lupus erythematosus (LE) is a multi-system disease, involving heart, kidney, central nervous system, skin and other organs.

MRL/Mp–lpr/lpr (MRL/lpr) mice are known to have an autosomal recessive mutant gene “lpr” which produces massive T cell proliferation, lupus nephritis and skin lesions which are similar to those of human LE.

We reported the findings of the relationship among skin lesion, proteinuria and lymph-node swelling in MRL mice and their hybrids.

MATERIALS AND METHODS

MRL/lpr, MRL/Mp–+/+ (MRL/n), F1 hybrid of MRL/lpr × MRL/n and F2 hybrid of F1 × F1 mice were maintained under specific pathogen free conditions. All examined mice were female and 5 months of age.

Lymph-node swelling, proteinuria and skin lesion were carefully checked, and histological studies of lymph-node, kidney and dorsal skin were performed in the mice examined.

Incidences and findings in the hybrid mice were compared to those of MRL/lpr mice.

![Graph showing lymph-node swelling in MRL mice and their hybrids at 5 months of age.](image)
Kanauchi H et al: Skin lesions in MRL hybrid mice.

RESULTS AND DISCUSSION

Incidences of lymph-node swelling were 84% in MRL/lpr mice, 0% in MRL/n mice, 0% in F1 hybrid mice and 21% in F2 hybrid mice (Fig. 1). Those of proteinuria were 91% in MRL/lpr mice, 8% in MRL/n mice, 7% in F1 hybrid mice and 31% in F2 hybrid mice (Fig. 2). Those of skin lesion were 70% in MRL/lpr mice, 0% in MRL/n mice, 0% in F1 hybrid mice and 7% in F2 hybrid mice (Fig. 3). The incidence of skin lesion in F2 hybrid mice was lower than the theoretical expected rate.

Histological findings of lymph-node, kidney and skin lesion in some F2 hybrid mice were similar to those in MRL/lpr mice. These findings suggested that the appearance of skin lesions in MRL mice is regulated not only by the lpr gene but also by other factors.

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REFERENCES

1. Murphy ED, Roths JB.: Genetic Control of Autoimmune Disease 1978;207-221.