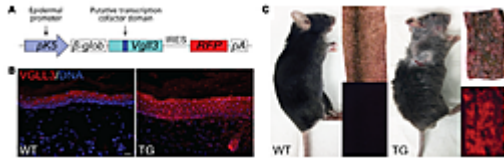




# Mouse Model of Autoimmunity

TECHNOLOGY NUMBER: 2019-174



## OVERVIEW

A transgenic mouse model that serves as a tool for studying autoimmune disease

- Provides insight into the female predilection for autoimmune diseases
- Offers a screening tool to develop therapies for diseases including lupus

## BACKGROUND

Autoimmune disease diagnoses include scleroderma, rheumatoid arthritis, and systemic lupus erythematosus. The precise causes for these illnesses are poorly understood, and their prevalence includes almost 8% of the US population. Cutaneous lupus can be associated with a wide range of dermatological manifestations. These skin pathologies may arise as presenting signs of systemic lupus, or they can occur on their own in the absence of any other disease manifestations. Research into the causes and treatment of autoimmune diseases have been proven to be difficult, so a need exists for an animal model that can offer important insights into the molecular mechanisms controlling these maladies.

## INNOVATION

Researchers at the University of Michigan have invented a transgenic mouse model for the study of autoimmune diseases. The model describes for the first time the role of a transcription factor named Vgll3 in the pathogenesis of autoimmune diseases. Vgll3 is enriched in the skin of females when compared to males, and its overexpression in mice leads to an autoimmune phenotype similar to lupus. This novel technology consists of a transgenic mouse model that permits upregulation of Vgll3 in the skin which produces a lupus-like skin rash in affected mice when compared to wild type animals. These visible and measurable skin abnormalities provide an easy means by which medical interventions can be screened and assessed for efficacy. This transgenic mouse model can also be modified and used to study other autoimmune disorders.

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## Category

Materials

Life Sciences

## Inventor

Andrzej Dlugosz

Joanne Kahlenberg

Johann Gudjonsson

Yun Liang

## Further information

Stefan Koehler

[shkohler@umich.edu](mailto:shkohler@umich.edu)

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## PATENT APPLICATION

Number: [WO2019136084A1](#)

### References

1. Billi, Allison C, et al. , The female-biased factor VGLL3 drives cutaneous and systemic autoimmunity