

BXSB/MpJ

Stock No: **000740**

 Recombinant Inbred (RI)

Live mice available in varying quantities. Ask Customer Service for details.

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BXSB mice develop a spontaneous autoimmune syndrome similar to lupus, which is accelerated in males (BXSB males have the mutant *Yaa*-containing Y chromosome). These mice may be useful in applications related to systemic lupus erythematosus.

Our preclinical efficacy testing services offer scientific expertise and an array of target-based and phenotype-based outcome measures, both in vivo and at endpoint, for flexible study designs and assay development in mouse models of Lupus. [See our full service platform.](#)

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GENETIC OVERVIEW

Genetic Background

Generation

[F169](#)

(2020-03-19 00:00:00)

[Yaa](#)

Allele Type

Spontaneous

Gene Symbol

Yaa

Gene Name

accelerated autoimmunity and lymphoproliferation transposition

V I E W G E N E T I C S

RESEARCH APPLICATIONS

Immunology, Inflammation and Autoimmunity Research

V I E W A L L R E S E A R C H A P P L I C A T I O N S

BASE PRICE

Starting at:

\$218.00 Domestic price for male 5-week

296.51 Domestic price for breeder pair

V I E W P R I C E L I S T

Details

Important Note

Males of this strain carry the BXSB-derived *Yaa* mutation on the Y chromosome.

Detailed Description

The Y-linked autoimmune accelerator locus (*Yaa*) on the Y chromosome of BXSB inbred male mice is the result of a duplication of an ~4 Mbp telomeric segment near the pseudoautosomal region of the X chromosome onto the Y chromosome. The duplicated segment contains 19 genes, including toll-like receptor 7 (*Tlr7*) and phosphoribosyl pyrophosphate synthetase 2 (*Prps2*). BXSB males have the mutant *Yaa*-containing Y chromosome, and are a model for a severe form of systemic lupus erythematosus (SLE). Their spontaneously arising autoimmune disease is characterized by the robust development of follicular T cells and germinal centers. Marginal zone B cells are depleted in BXSB-*Yaa* mice and a dramatic increase in number of peripheral monocytes is seen beginning at two months of age. Disease progression is characterized by lymph node and spleen enlargement, hemolytic anemia, hypergammaglobulinemia, anti-nuclear antibodies, and immune complex glomerulonephritis. This leads to morbidity on average of six months of age. BXSB females develop a greatly attenuated form of autoimmune disease; indicating that autoimmune disease acceleration is due to the presence of the *Yaa* mutation carried on the Y chromosome.

A duplicated copy of the *Tlr7* gene is primarily responsible for the autoimmune phenotype attributed to the *Yaa* mutation. The BXSB-*Yaa* mouse therefore provides a model for investigating the effects copy number variation of *Tlr7* on autoimmune disease. Therapeutic treatment of BXSB-*Yaa* mice with an anti-Type I interferon receptor antibody attenuates their autoimmune disease; indicating that Type I interferon signaling involved is in the pathogenesis of this disease.

The most suitable control animals are sex-matched, autoimmune disease-resistant BXSB.B6-*Yaa*⁺ consomic males carrying the C57BL/6-derived Y chromosome in place of the BXSB-derived mutant *Yaa*-containing Y chromosome. BXSB.B6-*Yaa*⁺ consomic mice (backcrossed to BXSB/MpJ inbred females for at least 40 generations) are described and available at The Jackson Laboratory as Stock No. [021330](#).

A note on outcrossing BXSB males with other inbred females: similar autoimmune disease acceleration occurs in males (but not in females) from an outcross of BXSB males to NZB females. Acceleration does not occur in offspring of the reciprocal cross. The same effect of *Yaa* is seen in offspring of outcrosses to strains SJL, C57BL/6, and AKR.

In addition, BXSB males lacking the interleukin 21 receptor (BXSB-*Yaa*//*Il21*^{r^{-/-}} or BXSB-*Yaa*//*Il21*^{-/-}) are highly resistant to disease and are therefore a model for investigating the role of interleukin 21 in disease pathogenesis. Populations of regulatory CD8⁺ and potentially natural killer cells are also actively retarding disease in this model; making them useful to investigate the roles of such regulatory cells in autoimmune disease.

+ Control Suggestions

+ Selected References

- Genetics

+ Yaa

- Disease/Phenotype

+ Disease Terms

+ Research Areas By Phenotype

+ Mammalian Phenotype Terms by Genotype

+ Phenotype Information

+ References

- Technical Support

Genotyping Protocols

QPCR: [Yaa Alternate1](#)

QPCR: [X chrom qPCR](#)

[Genotyping resources and troubleshooting](#)

Dietary Information

LabDiet® 5K52 formulation (6% fat)

Breeding Considerations

This is an inbred strain. Males carry the BXSB-derived *Yaa* mutation on the Y chromosome. To maintain the live inbred colony, unaffected females (+/+) may be bred with affected males (*Yaa/Y*).

Additional Breeding and Husbandry Support

Mating System

+/+ sibling x Hemizygote

Appearance

white-bellied agouti, affected

Related Genotype: $A^W/A^W X/Yaa$ (male)

white-bellied agouti, unaffected

Related Genotype: $A^W/A^W X/X$ (female)

Citation

When using the BXSB/MpJ mouse strain in a publication, please [cite the originating article\(s\)](#) and include JAX stock #000740 in your Materials and Methods section.

Animal Health Reports

[Facility Barrier Level Descriptions](#)

 [AX10 \(Standard\)](#)

🔵 Pricing & Availability



Live mice available in varying quantities. Ask Customer Service for details.

Available

Domestic International

Pricing effective for USA, Canada and Mexico shipping destinations

LIVE MOUSE

AGE	SEX	GENOTYPE	PRICE
5 weeks	Female	Wildtype for Yaa	\$78.51
5 weeks	Male	Hemizygous for Yaa	\$218.00
6 weeks	Female	Wildtype for Yaa	\$78.51

6 weeks	SEX	Hemizygous for Yaa	\$218.00
7 weeks	Female	Wildtype for Yaa	\$78.51
7 weeks	Male	Hemizygous for Yaa	\$218.00
8 weeks	Female	Wildtype for Yaa	\$78.51
8 weeks	Male	Hemizygous for Yaa	\$218.00
9 weeks	Female	Wildtype for Yaa	\$78.51
9 weeks	Male	Hemizygous for Yaa	\$218.00

BREEDER PAIR		PRICE
SEX	GENOTYPE	
Female	Wildtype for Yaa	\$296.51
Male	Hemizygous for Yaa	

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