B6.129P2-Nos2<sup>tm1Lau</sup>/J

Stock No: 002609 | iNOS-

- Congenic, Targeted Mutation

AVAILABLE NOW

PLACE ORDER

Sized to accommodate orders of up to 10 or more with age range. Ask Customer Service for details.

Overview
Also Known As: iNOS-

$\textit{Nos2}^{\text{tm1Lau}}$ homozygotes, unlike $\textit{Nos2}$ wildtype mice, have virtually no serum nitric oxide response, but were susceptible to LPS-induced death. $\textit{Nos2}^{\text{tm1Lau}}$ homozygotes exhibit altered responses to $M. \text{bovis}$ (BCG), systemic $E. \text{coli}$ infection, $M. \text{tuberculosis}$ and $M. \text{pulmonis}$. In addition, wound healing properties of fibroblasts are impaired in $\textit{Nos2}^{\text{tm1Lau}}$ homozygotes. These mice may be suitable for use in studies of inflammatory conditions including rheumatoid arthritis, inflammatory bowel disease, cardiac allograft rejection, hepatotoxicity, myocardial ischemia-reperfusion and septic shock.

Donating Investigator

Dr. Victor Laubach, University of Virginia Health Sci. Ctr.
Mice homozygous for the \( \text{Nos2}^{\text{tm1Lau}} \) targeted mutation resemble wildtype mice in appearance and histology. Homozygotes are viable and fertile. Unlike \( \text{Nos1} \) and \( \text{Nos3} \), \( \text{Nos2} \) is synthesized \textit{de novo} in response to a variety of inflammatory stimuli. Induction of \( \text{Nos2} \) results in the production of large amounts of nitric oxide (NO) over prolonged periods of time. Excessive NO production has been shown to be beneficial through its antitumor and antimicrobial activities. It is also thought to cause tissue damage and contribute to pathology in a variety of inflammatory conditions including rheumatoid arthritis, inflammatory bowel disease, cardiac allograft rejection, hepatoxicity, myocardial ischemia-reperfusion and septic shock. NO has been demonstrated to play a role in the regulation of blood pressure and hemodynamics. In an LPS-induced model of septic shock, \( \text{Nos2}^{\text{tm1Lau}} \) homozygotes had virtually no serum NO response, but were susceptible to LPS-induced death. \( \text{Nos2}^{\text{tm1Lau}} \) homozygotes exhibit altered responses to \( \text{M. bovis} \) (BCG), systemic \( \text{E. coli} \) infection, \( \text{M. tuberculosis} \) and \( \text{M. pulmonis} \). In addition, wound healing properties of fibroblasts are impaired in \( \text{Nos2}^{\text{tm1Lau}} \) homozygotes. Also known as iNOS.
Genotyping Protocols
Standard PCR: Nos2<sup>tm1Lau</sup>

Genotyping resources and troubleshooting

Dietary Information
LabDiet® 5K52 formulation (6% fat)

Breeding Considerations

This strain is a good breeder.

Additional Breeding and Husbandry Support

Mating System
Homozygote x Homozygote

Appearance
black

Related Genotype: a/a

Citation

When using the iNOS mouse strain in a publication, please cite the originating article(s) and include JAX stock #002609 in your Materials and Methods section.

Animal Health Reports

Facility Barrier Level Descriptions

Pricing & Availability

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<td>Homozygous for Nos2&lt;sup&gt;tm1Lau&lt;/sup&gt;</td>
<td>$131.39</td>
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