B6.Cg-Tg(Myh6-tTA)6Smbf Tg(tetO/CMV-ATP2B4,-lacZ)1Hu sa/Mmjax

MMRRC Stock No: 43951-JAX | huPMCA4b Tet-Off

Congenic, Transgenic

AVAILABLE

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Also Known As: huPMCA4b Tet-Off, BT:tTA+/hPMCA4b+

TtA+/hPMCA4b bi-transgenic mice allow targeted overexpression or Tet-Off conditional expression of human ATP2B4 (or PMCA4b) in cardiomyocytes. Overexpression of ATP2B4 has a cardioprotective effect following ischemic injury. These mice are suitable for use in applications related to the study of heart failure.

Donating Investigator
Mansoor Husain, Toronto General Hospital Research Instt.
Hemizygous bi-transgenic tTA+/hPMCA4b mice are viable and fertile and harbor two transgenes using the Tet-Off system. The tTA transgene uses a rat α-MHC (Myh6 - myosin, heavy polypeptide 6, cardiac muscle, alpha) promoter directing expression of the transcriptional transactivator (tTA) to cardiomyocytes. The hPMCA4b transgene has ATP2B4 cDNA linked to a bi-directional viral CMV promoter containing Tet Operator sites. lacZ cDNA is present on the other side of the bi-directional CMV promoter. X-gal staining and immunohistochemistry confirm the presence of nuclear-localized lacZ in cardiomyocytes. RT-PCR confirms expression of ATP2B4 in heart, but not in liver, kidney, lung, brain intestine, muscle or aorta or in DOX-treated littermates. ATP2B4 is involved in intracellular calcium homeostasis; heart failure is associated with reduced expression of plasma membrane ATP2B4. In the absence of tetracycline (or its analog doxycycline (dox)), the promoter limits hPMCA4b protein expression to cardiomyocytes. Overexpression results in elevated blood pressure, increased cardiac contractility and lusitropy (rate of myocardial relaxation), as well as an inhibition of cardiac NOS and nNOS-specific activities. Prolonged mild cardiac hypertrophy does not result in cardiac dysfunction. Following myocardial ischemia reperfusion injury (IRI), PMCA4b overexpression preserves cardiac function, limiting infarct progression, cardiac hypertrophy and heart failure (HF). Because this binary transgenic system also allows for a second level of control (i.e., addition of dox), expression of the hPMCA4b protein can be completely abolished; reversing the phenotype.
Genotyping Protocols
Standard PCR: Tg(tetO/CMV-ATP2B4,-lacZ)1Husa
Standard PCR: Tg(Myh6-tTA)6Smbf

Genotyping resources and troubleshooting

Dietary Information
New Diet as of March 2015: Lab Diet® 5K0Q (6% fat)

Breeding Considerations
When maintaining a live colony, mice that are hemizygous for both alleles may be bred together, to wildtype mice from the colony or to C57BL/6J inbred mice (Stock No. 000664). The donating investigator has not attempted to make either allele homozygous.

Additional Breeding and Husbandry Support

Mating System
See "Breeding Considerations"
Maintained as Hemizygous for Tg(Myh6-tTA), Hemizygous for Tg(tetO/CMV-ATP2B4,-lacZ) x Noncarrier for Tg(Myh6-tTA), Noncarrier for Tg(tetO/CMV-ATP2B4,-lacZ) OR Noncarrier for Tg(Myh6-tTA), Noncarrier for Tg(tetO/CMV-ATP2B4,-lacZ) x Hemizygous for Tg(Myh6-tTA), Hemizygous for Tg(tetO/CMV-ATP2B4,-lacZ). C57BL/6J can be used in place of Noncarrier for Tg(Myh6-tTA), Noncarrier for Tg(tetO/CMV-ATP2B4,-lacZ) for breeding.

Citation
When using the huPMCA4b Tet-Off mouse strain in a publication, please cite the originating article(s) and include MMRRC stock #43951 in your Materials and Methods section.

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