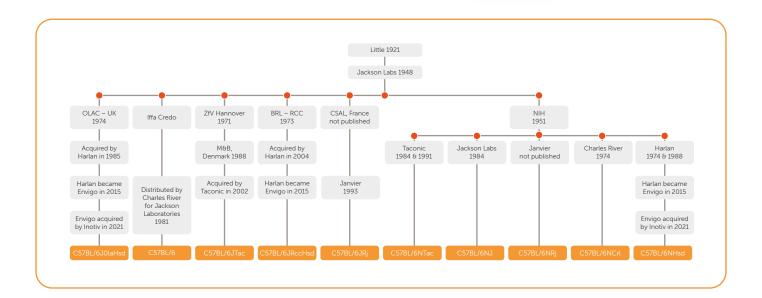




C57BL/6

Substrain information





RESEARCH USES

- Background for induced and genetically modified models
- Diet-Induced Obesity
- Toxicology
- Cardiovascular
- Superovulation

- Immunology
- Aging
- Drug Addiction
- Alcoholism
- General Purpose

GENERAL CHARACTERISTICS

	C57BL/6∩Hsd	C57BL/6JOlaHsd	C57BL/6JRccHsd			
Origins	Originally developed by Little in 1921; to the Jackson Laboratory, Bar Harbor, Maine, 1946					
	to National Institutes of Health (NIH), Bethesda, Maryland in 1951; to Harlan Laboratories in 1988. Harlan became Envigo in 2015, then Envigo was acquired by Inotiv in 2021.	to Laboratory Animal Centre United Kingdom, in 1974; to Harlan Laboratories in 1985. Harlan became Envigo in 2015, then Envigo was acquired by Inotiv in 2021.	to the Jackson Laboratory, Bar Harbor, Maine, 1946; to Rcc, Fullinsdorf, Switzerland, in 1973; to Harlan Laboratories in 2004 Harlan became Envigo in 2015, then Envigo was acquired by Inotiv in 2021.			
Litter Average	6.0					
Common Characteristics	Low tumor incidence, high preference for alcohol, micropthalmia, hydrocephalic, age-related hearing loss, alopecia					
Coat Color	a/a; black					
Haplotype	H1 ^c , H-2 ^b , H-3 ^a					
Common Genetic Characteristics	Thy1.2, Cdh23 ^{Ahi} , Ahr ^b , Apoa-1 ^a , Car-2 ^a , Es-1 ^a , Es-2 ^b , Es-3 ^a , Gpd-1 ^a , Gpi-1 ^b , Hbbs, Idh-1 ^a , Ldr-1 ^a , Mod-1 ^b , Pep-3 ^a , Pgm-1 ^a , Tr ^{fb} , Ptprc ^a					
Additional Notes	Although there is some anecdotal information on different research uses and subline differences in performance for specific model, there is relatively little published information in this area. Much of this information is complicated by improper identification of sources and strain/subline nomenclature in many peer-reviewed publications. We have no evidence that most of these sublines are not interchangeable, unless published or internal data has demonstrated an important difference in response that affects the interpretation of experimental results.					

SUBSTRAIN GENE MUTATIONS

STRAIN	SUPPLIER .	DELETION			
		∩nt	Scna	Mora1	Rd8
C57BL/6JOlaHsd	Inotiv	No	Yes	Yes	No
C57BL/6JRccHsd	Inotiv	No	No	No	No
C57BL/6NHsd	Inotiv	No	No	No	Yes
C57BL/6J	Jackson Laboratory	Yes	No	No	No
C57BL/6ByJ	Jackson Laboratory	No	No	No	No
C57BL/6J	Charles River	Yes	No	No	No
C57BL/6JCrl	Charles River	Yes	No	No	No
C57BL/6NCrl	Charles River	No	No	No	Yes
C57BL/6JBomTac	Taconic	No	No	No	No
C57BL/6NTac	Taconic	No	No	No	Yes
C57BL/6JRj	Janvier	Not published	Not published	Not published	Not published
C57BL/6JRj	Janvier	Not published	Not published	Not published	Not published

IMPACT OF GENETICS ON RESEARCH

Nnt = nicotinamide nucleotide transhydrogenase; this gene encodes an integral protein of the inner mitochondrial membrane. The enzyme couples hydride transfer between NAD(H) and NADP(+) to proton translocation across the inner mitochondrial membrane.

Scna = alpha synuclein; one in a family of structurally related proteins that are prominently expressed in the brain, particularly in areas associated with learning and adaption. The exact function of alpha synuclein is not yet known.

Mnrn1 = multimerin 1; multimerin 1 is a stored platelet and endothelial cell adhesive protein that shows significant conservation. In vitro, multimerin 1 supports platelet adhesion and it also binds to collagen and enhances von Willebrand factor-dependent platelet adhesion to collagen.

Rd8 = retinal degeneration 8; the rd-8 mutation is due to a single base pair mutation in the CRB1 gene. This gene when mutated in humans is linked to macular degeneration and other age-related vision loss. Mice with this mutation are nearly blind by the time they are 8 weeks of age.

Nicotinamide nucleotide transhydrogenase

The absence of the NNT protein has been associated with impaired glucose homeostasis control and reduced insulin secretion and is also required for normal mitochondrial function including metabolism and protection from oxidative stress (Huang et al, 2006).

Alpha synuclein

Although C57BL/6JOlaHsd mice have a loss-of-function deletion in the Scna gene, they display no up regulation of beta-synuclein or gamma-synuclein and the expression of synphilin-1 is unaffected. Spatial learning also seems to be unaffected (Specht, 2001).

Multimerin 1

C57BL/6JOlaHsd mice display impaired platelet adhesion and impaired thrombus formation that can be rescued by a functioning copy of multimerin-1 (Reheman *et al.*, 2010).

Rd8

The rd8 mutation is a single nucleotide deletion in the Crb1 gene, which results in a form of retinal degeneration appearing with distinct clinical appearance including multiple light-colored spots in the fundus of the eye that correspond histologically to retinal folds, pseudorosettes, and focal retinal dysplasia and degeneration (Chang et al, 2002).

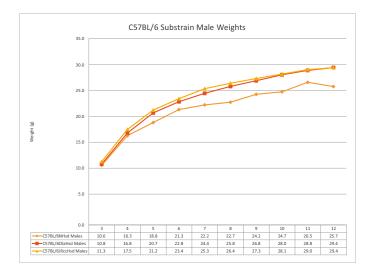
GENETIC CONCORDANCE AMONG C57BL/6 SUBSTRAINS

	C57BL/6J	C57BL/6JRCCHSD	C57BL/6JOLAHSD	C57BL/6NHSD	C57BL/6NTAC	C57BL/6NCRL
C57BL/6J		98.5%	98.5%	97.8%	97.8%	97.8%
C57BL/6JRccHsd	98.5%		No	99.3%	99.3%	99.3%
C57BL/6JOlaHsd	98.5%	100%		99.3%	99.3%	99.3%
C57BL/6NHsd	97.8%	99.3%	99.3%		100%	100%
C57BL/6NTac	97.8%	99.3%	99.3%	100%		100%
C57BL/6NCrl	97.8%	99.3%	99.3%	100%	100%	

Genetic drift is the change in frequency in which a gene appears in a population, through mutation, regardless of the adaptive value of the mutation. In an inbred population, natural random mutation occurs rather infrequently. Genetic drift is a normal process for any breeding population and thus cannot be prevented. It can only be slowed through various breeding and cryopreservation techniques. Most random mutations in a populations are single nucleotide polymorphisms and do not affect phenotype due the redundancy of the genetic code. Inotiv utilizes a single source for all inbred populations of the same strain, a common parent rule to prevent subline divergence, and we are in the process of developing a global breeding and cryopreservation program for all C57BL/6 substrains worldwide. The single nucleotide polymorphism (SNP) panel for the above concordance table contained 560 SNP's. For more information regarding SNP testing of our C57BL/6 substrains or our global breeding and cryopreservation program, please contact Inotiv's Veterinary Science, Research and Support Team at RMSTechnicalServices.na@inotivco.com.

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GENETIC CONCORDANCE AMONG C57BL/6 SUBSTRAINS





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