CE/J mice are very responsive to phytohemagglutinin and resistant to amyloid induction and development of amyloidosis. CE/J mice express a single isoform of SAA designated SAA2.2, which is structurally unable to form amyloid fibrils. Viral expression of amyloidogenic proteins in CE/J mice does result in amyloid deposition, making this a useful model to examine the amyloid deposition potential of altered SAA proteins. These mice have a high incidence of ovarian tumors and hepatomas.
Important Note
This strain is homozygous for Cdh23<sup>ahl</sup>, the age related hearing loss 1 mutation, which on this background results in progressive hearing loss with onset after 10 months of age. Of note, most mice exhibit a small ear phenotype; the cause has not been determined.

Detailed Description
CE/J mice are very responsive to phytohemagglutinin and resistant to amyloid induction and development of amyloidosis. Amyloidosis is the extracellular deposition of the acute phase protein 'serum amyloid A' (SAA), which is induced in response to inflammation. CE/J mice express a single isoform of SAA designated SAA2.2, which is structurally unable to form amyloid fibrils. Viral expression of amyloidogenic proteins in CE/J mice does result in amyloid deposition, making this a useful model to examine the amyloid deposition potential of altered SAA proteins.

Early studies by Dr. Margaret Dickie reported that approximately 1/3 of CE females and of reciprocal DBA x CE F1 hybrids were found to have ovarian tumors at 20 months of age. Virgin reciprocal DBA x CE F1 hybrids were found to develop hyperplasia of the uterus and an increased uterine sensitivity to estrogen that increases with age. This pathology results from a hormonal imbalance characteristic of this F1 cross and breeding the female can ameliorate this trait.

It has also been reported that gonadectomized CE females and males develop adrenal cortical tumors by 6 and 12 months of age, respectively. Adrenal cortical tumorogenesis appeared to be followed by development of secondary sex organs in these gonadectomized mice. Basophile pituitary tumors were also found after 14 months of age in gonadectomized CE mice and F1 hybrids of CE and DBA or C57. The highest incidence was seen in reciprocal CE x DBA F1 hybrids.

Another early study reported that an increase in diet fat content from 4% to 11% strongly enhanced hepatocarcinogenesis in the male F1 hybrid offspring of CE/J with DBA/1J or DBA/2J, or C3H/He but not C57BL/6J. Subsequently, in 1971 Festing and Blackmore published the results of a life span and spontaneous disease profile of 15 inbred mice housed under specific-pathogen-free conditions. Autopsy results of 40 CE mice revealed a high incidence of hepatomas, but no other significant finding.

Development

Selected References

Genetics

Il3ra<sup>m1</sup>

Hc<sup>0</sup>
Cdh23<sup>ahl</sup>

Mx<sup>1</sup><sup>as2</sup>

Ah<sup>b-2</sup>

Tyr<sup>c-e</sup>

**Disease/Phenotype**

**Disease Terms**

**Research Areas By Genotype**

**Mammalian Phenotype Terms by Genotype**

**Phenotype Information**

**References**

**Technical Support**

**Genotyping Protocols**
Genotyping resources and troubleshooting

**Appearance**
greyish white

**Related Genotype:** <sup>A</sup>W/<sup>A</sup>W <sup>Tyr</sup><sup>c-e</sup>/<sup>Tyr</sup><sup>c-e</sup>

**Citation**
When using the CE/J mouse strain in a publication, please include JAX stock #000657 in your Materials and Methods section.

**Animal Health Reports**
Production of mice from cryopreserved embryos or sperm occurs in a maximum barrier room, <sup>G</sup>200

**Pricing & Availability**

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