Transgenic Animal Models for Brain 5-HT Deficiency: An Editorial Summary

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Editorial

Animal: Pet-1 KO mice
First Report Year: 2003
Methods: Pet-1 Knock out
Lab: Evan Deneris
Loss of 5-HT Neurons: 80% deficiency of the central 5-HT neurons
Life Span and Body Weight: Survive to adult and normal body weight

Behavior:
• Heightened anxiety-like and aggressive behavior in adults [1].
• Extended and exacerbated period of breathing instability that occurs immediately after birth during which respiratory homeostasis is vulnerable to environmental challenges [2].
• Abnormal maternal behavior which affects offspring survival [3].
• A lack of cognitive deficits and an anxiety phenotype complicated by hypoactivity and defensiveness [4].
• Autoresuscitation responses to hypoxia-induced apnea are delayed in newborn mice [5].
• Altered ventilatory and thermoregulatory control [6].
• Bradycardia (the first 2 postnatal weeks) [7].
• Failed heart rate recovery at early age exposed to episodic anoxia [8].
• Autonomic dysregulation during mild cold stress in the neonatal period [9].

Animal: Lmx1b conditional KO mice
First Report Year: 2006
Methods: Conditional Lmx1b Knock out
Labs: Zhou-Feng Chen, Yu-Qiang Ding and Lin Xu.

Loss of 5-HT Neurons: The initial generation of central 5-HT neurons appeared normal (at E11). However, the expression of both 5-HT-specific and non-5-HT-specific markers was lost in these neurons at later stages of development. Almost all central 5-HT neurons failed to survive.

Life Span and Body Weight: Survive to adult, growth retardation [18,19] and persistent leanness [18].

Behavior:
• More extended daytime sleep, suppressed respiration, altered body temperature control, and decreased blood pressure (BP) and heart rate (HR) during nighttime; Tph2 ko females, exhibit impaired maternal care leading to poor survival of their pups [19].
• Exaggerated aggression and decreased anxiety [20].
• A severe low bone mass phenotype affecting axial (vertebrae) and appendicular (long bones) skeleton while bone length and width were unaffected [21].
• Respond to METH in the same manner as wild-type controls, despite showing enhanced drug-induced hyperthermia [22].
• Substantial deficits in numerous validated tests of social interaction and communication; highly repetitive and compulsive behaviors; Newborn mice show delays in the

Animal: Tph2 KO mice
First Report Year: 2008 [17]
Methods: Tph2 Knock out
Labs: Katerina Savelieva, Klaus-Peter Lesch, Michael Bader, Gerard Karsenty, Donald M. Kuhn and Yi Rao.

Life Span and Body Weight: Survive to adult, growth retardation [18,19] and persistent leanness [18].

Behavior:
• Less sensitive to mechanical stimuli and exhibited enhanced inflammatory pain, the analgesic effect of several antidepressant drugs, including selective serotonin reuptake inhibitors (SSRIs), serotonin-norepinephrine reuptake inhibitors (SNRIs), and tricyclic antidepressants, was either abolished or greatly attenuated [14].
• Increase in wakefulness [15].
• Enhanced contextual fear memory [16].

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Received January 02, 2013; Accepted January 05, 2013; Published January 07, 2013


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expression of key developmental milestones and their diminished preference for maternal scents over the scent of an unrelated female is a forerunner of more severe socialization deficits that emerge in weanlings and persist into adulthood [23].

- Males did not show a preference between male and female bedding and genital odour [24].

References


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