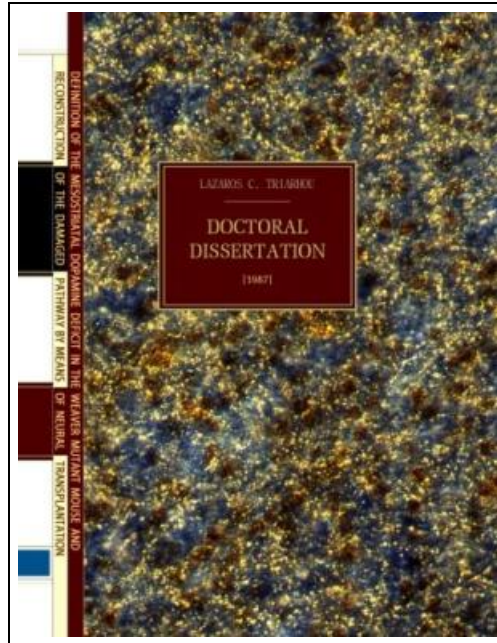


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Createspace Independent Publishing Platform, United States, 2016. Paperback. Book Condition: New. 246 x 189 mm. Language: English . Brand New Book \*\*\*\*\* Print on Demand \*\*\*\*\*.Striatal dopamine (DA) deficiency has been recognized as one of the phenotypic expressions of the mutant gene in homozygous weaver mice. The present study has been aimed at (i) characterizing the deficit of the mesostriatal pathway in the weaver mutant in relation to the number of DA neurons in the ventral mesencephalon and to the synaptology of DA containing nerve terminals in the striatum, and (ii) attempting to reconstruct the anatomical deficit by grafting ventral mesencephalic anlagen to the weaver striatum. Experimental procedures included dissection of embryonic brain, intracerebral grafting, light and electron microscopic immunocytochemistry, and quantitative morphometry. The results show that the weaver mutant mouse is characterized by a spontaneous loss of DA neurons in all mesencephalic DA cell groups. The available anatomical evidence suggests that the mesostriatal DA projection does not reach full development and further regresses with age. Despite the naturally occurring degenerative process, the striatum of weaver mutants is receptive to reinnervation by DA afferent fibers originating in grafts from normal donor embryos. The synaptic investment of striatal cellular domains approximately resembles that seen under normal circumstances. This investigation is relevant to the understanding of (i) pathogenetic mechanisms associated with the mesostriatal DA deficiency found in familial Parkinsons disease, and (ii) modalities of therapeutic intervention in abiotrophic diseases of the brain.

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