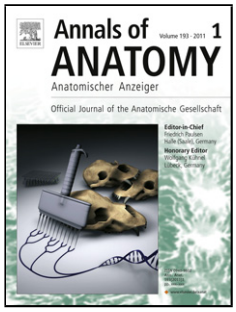

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PII: S0940-9602(17)30052-3
DOI: http://dx.doi.org/doi:10.1016/j.aanat.2017.05.001
Reference: AANAT 51156

To appear in:

Received date: 9-9-2016
Revised date: 29-4-2017
Accepted date: 11-5-2017

Please cite this article as: Nooh, Hanaa Z., El-Saify, NerGhada H., Eldien, Nermeen M.Noor, Neuroprotective effects of food restriction on autonomic innervation of the lacrimal gland in rat.Annals of Anatomy http://dx.doi.org/10.1016/j.aanat.2017.05.001

Neuroprotective effects of food restriction on autonomic innervation of the lacrimal gland in rat

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Abstract

Inflammatory mechanisms and oxidative stress play important roles in age-related lacrimal gland (LG) degeneration as well as neural degeneration. Research suggests that caloric restriction can prevent age-related LG dysfunction and increase the life span of neurons. In the present study, we hypothesized that caloric restriction prevents age-related LG dysfunction by ameliorating the influence of inflammatory/oxidative stress on autonomic neurons controlling lacrimal function. We evaluated the effects of food restriction (FR) on inflammatory/oxidative status and on autonomic neural/neuroglial cell populations in LGs from aging rats. A total of 45 female albino rats were divided into young adult, aged, and aged-FR groups. The FR group was subjected to a 50% reduction in food from 14–20 months of age. LG samples were collected for each group and subjected to biochemical, histological, and immunohistochemical studies. LGs from aged-FR rats, rather than those from aged rats, showed preservation of their cellular structures, organelles, and Schwan cell units. LG preservation was associated with a marked decrease in inflammatory markers, an increase in cellular antioxidants, and the up-regulation of choline acetyltransverase, tyrosine hydroxylase, neuron-specific enolase and S100. These findings strongly suggest that in aged rats, both oxidative and inflammatory stressors directly contribute to LG dysfunction by mediating the degeneration of autonomic neurons, and that FR can protect against these effects.

Keyword, lacrimal gland, food restriction, inflammatory/oxidative stress, autonomic neurons

1. Introduction

Aging encompasses the biological and physiological changes that occur throughout life. The effects of aging include reduced body resistance to stress and increased vulnerability to disease (Shock et al. 2015). In the absence of malnutrition, food restriction (FR) has shown several effects retarding the aging process in rodents, monkeys and humans (Talhati et al., 2014). Moreover, it is believed that FR-induced decreases in oxidative stress are responsible for these effects in rats and mice (Walsh et al. 2014).

Oxidative stress and associated inflammation are believed to initiate the development of dry eye disease, which is an age-related functional decline of tear production from the LG (Uchino et al. 2012). Oxidative damage to the LG increases with age, which results in the progressive loss of functional efficiency of various cellular processes (Batista et al., 2012). Age-related increases in oxidative stress in