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Microglial alterations in rd10 retina: effect of progesterone

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Abstract

Purpose: Inflammation is related many retinal diseases pathogenesis, such as retinitis pigmentosa (RP), age-related macular degeneration (AMD), diabetic retinopathy, etc. RP patients present cells of the immune system and inflammatory factors in the subretinal space, as well as in the vitreous and aqueous humor, due to the alteration in the retinal pigment epithelium (RPE). However, the importance of this inflammation is not completely known.

Our group has previously shown that progesterone delays photoreceptor cell death in the rd1 mice, a RP experimental model. In the brain, progesterone is able to: i) decrease lipid peroxidation and oxidative stress; ii) limit the release of inflammatory cytokines and; iii) decrease glutamate toxicity.

The purpose of this work was to demonstrate if progesterone is able not only to increase photoreceptor survival but also to ameliorate inflammation effects in the retina of a different RP animal model, the rd10 mice.

Methods: Animals were treated in accordance to the ARVO statement for the use of animals in ophthalmic and vision research. 50, 100, 150 or 200 mg/Kg of progesterone were administered orally to rd10 mice, at post-natal days 15, 17 and 19. Mice were euthanized at post-natal day 21 and survival of photoreceptors was examined in three retinal areas (central, mid and peripheral retina) by histochemistry. Progesterone effect on microglial activation and nitric oxide release was determined by iba-1 immunohistochemistry and western-blot. Finally, glutamate concentrations in rd10 mice retinas with and without progesterone treatment were studied by high pressure liquid chromatography (HPLC).

Results: Progesterone increased photoreceptor survival in rd10 mice and a positive and significant correlation between progesterone dose and number of photoreceptor rows was established in the three retinal areas studied. An increase in iba-1 positive cells was found in rd10 retinas and progesterone was able to decrease not only the number of these cells but also their migration to the damage area. We demonstrate a decrease in iNOS (inducible nitric oxide synthase) and an increase in glutamate concentrations in rd10 retinas that progesterone was not able to modify.

Conclusions: Progesterone may be used to ameliorate retinal microglial activation in RP and in other retinal diseases that have been related to inflammation.

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