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OPHTHALMOLOGY
Editorial

Aqueous Deficiency Dry Eye – Current and Future Treatment Paradigms

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Received: September 30, 2015; **Published:** September 30, 2015

Aqueous Deficiency Dry Eye (ADDE) is a complex disease, which targets ocular surface and tear film causing abnormal tear production. Most common symptoms of dry eye include a frequent scratching or stinging sensation in the eye, blurred vision, sensitivity to light, redness, difficulty in night driving and wearing contact lenses thereby severely hampering normal vision and affecting the quality of life. The disease affects over 10 million Americans across all races and ethnicities resulting in progressively high health care costs each year. Clinical data obtained from ADDE patients show that women are significantly more likely to suffer from dry eye compared to men. Interestingly, the current treatment available in the market is very limited. Immunosuppressive drugs and artificial tear therapy are primarily prescribed for ADDE. However, these treatment options offer temporary relief and are mostly symptomatic. Additionally, long term use of immunosuppressive drugs like cyclosporine A or corticosteroids have severe side effects. Thus unraveling the pathophysiology of ADDE might not only further our current understanding of the disease and also allow us to come up with alternative therapies which address the cause of the disease.

The current treatment options for ADDE primarily focus on inflammation. Indeed, several studies have elucidated T lymphocyte mediated inflammation in lacrimal glands. This phenomenon occurs via a feed forward mechanism where T-cell activation leads to elevation of cytokine levels thereby causing more damage to the local tissue resulting in tissue atrophy. Lymphocytic infiltration and tissue atrophy in animal models of ADDE have been extensively demonstrated. However, inflammation might not be the first step in development and progression of ADDE. It is plausible that glandular dysfunction precedes cellular inflammation during development of ADDE. In the recent decade, a number of studies have hinted at glandular dysfunction to be the first step towards development of ADDE.

Lacrimal glands produce tear proteins and fluid via a plethora of cellular signaling pathways. Majority of the signaling output is based on the neural input and is primarily calcium dependent. Protein and fluid production in the lacrimal gland is tightly regulated and occurs in a concerted fashion via these signaling cascades. Moreover additional adjustments occur via crosstalk between these signaling pathways. Any alteration in such an elaborate and extensive network can affect protein and fluid production. This in turn can affect overall tear protein composition as well as tear volume and osmolarity. Such signaling defects in lacrimal glands are capable of inducing local immune cells to secrete proinflammatory cytokines resulting in lymphocytic infiltration thereby initiating a full scale immune response. Early detection and diagnosis is essential for management of ADDE. Thus biomarkers for lacrimal gland dysfunction might serve as early diagnostic tools for ADDE. Recently, a study showed that functional restoration of lacrimal glands was possible by bioengineered organ replacement. Moreover, the presence of stem-like cells in lacrimal glands has also been reported. Thus studies focused on reconstruction of the glandular architecture and restoration of lacrimal gland function might serve as a defining moment in treatment of ADDE.

Volume 2 Issue 2 September 2015

Citation: Sumit Bhattacharya. "Aqueous Deficiency Dry Eye–Current and Future Treatment Paradigms". *EC Ophthalmology* 2.2 (2015):96.