The lacrimal gland is the primary source for aqueous portion of the tear film. This portion contains water, electrolytes and proteins, which are necessary for the health and the maintenance of the cells of the ocular surface. Noradrenaline (NA), released from sympathetic nerves, is a major stimulus of lacrimal gland secretion. Here, lacrimal gland acinar cells response to adrenergic receptors activation were examined, with special reference to intracellular Ca2+ concentration ([Ca2+]i) dynamics.

In the present study, detection of mRNA of acinar cells specific to adrenergic receptor subtypes was determined by RT-PCR. All kinds of adrenergic receptors were detected except α2c and β1 in acinar cells of lacrimal glands. NA (30 μM) induced an increase in [Ca2+]i in acinar cells. NA-induced [Ca2+]i changes showed a biphasic behavior; the first step involved a steep phase of rapidly increasing [Ca2+]i, followed by the second plateau phase step. The removal of extracellular Ca2+ and the use of Ca2+ channel blockers did not completely inhibit the NA-induced [Ca2+]i increases. This reaction did not persist for long and the second plateau phase disappeared. Furthermore, suramin (a G protein antagonist) inhibited these increases. Phenylephrine (an α1 adrenoceptor agonist) induced a strong increase in [Ca2+]i. However, clonidine (an α2 adrenoceptor agonist) and isoproterenol (a β adrenoceptor agonist) failed to induce a [Ca2+]i increase. These findings indicated that NA activation resulted primarily in Ca2+ mobilization from intracellular Ca2+ stores and that NA activates α1 adrenoceptors which cause an increase in [Ca2+]i by production of IP3. Our results suggested that α1 adrenoceptors were key receptors in calcium-related cell homeostasis and exoclines in lacrimal glands.