Flux change in basophil membrane is not the main pathogenesis for hypersensitivity

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Abstract: The oxidation process is one of the most important natural processes. Oxidative change in hypersensitivity is believed to be an important process in the pathogenesis. However, the clear explanation on the transmembrane flux change of basophil and its correlation to hypersensitivity pathogenesis has never been reported. Here, the author determines the transmembrane oxidation flux in basophil. The simulation test to determine the oxidation flux change based on nanomedicine technique is used. Of interest, no change of flux can be detected. Therefore, this work can support the finding that the oxidation flux change is not an important part in the pathogenesis of basophil-related hypersensitivity.

Keywords: oxidation, basophil, flux, nanomedicine

Introduction

Basophil has a significant role in hypersensivity. Calcium mobilization is differentially involved in signaling to chemoattractants in basophils and that it is correlated with the agonist's efficacy to induce mediator release (Heinemann et al 2003). Synthesis of leukotriene (LT) C 4 by basophils and mast cells is an important component of IgE-mediated inflammation, resulting in increased levels of the cysteinyl leukotrienes (cysLTs) LTC 4 , LTD 4 , and LTE 4 (Gauvreau et al 2005). In basophils, under current-clamp conditions, spontaneous fluctuation of zero-current potentials is clearly apparent, presumably due to the activities of some ion channels generating a small amount of current flux through the membranes of these cells (Kawa 1989). Oshiro et al (1997) found that that certain G protein(s) promoted Ca^{2+}-dependent exocytosis in human basophils.

The query whether the property of the membrane influences basophile activation is of interest. However, the clear explanation on the transmembrane flux change of basophil and its correlation to hypersensitivity pathogenesis has never been reported. Basically, transmembrane flux means the changes in ion movement across the plasma membrane. For general biological cell, calcium ion change is corresponding to most of ion flux change. Previously, study of transmembrane flux is very difficult. Fortunately, the new development in nanotechnology, it is more feasible to determine the transmembrane flux. Here, the author determines the transmembrane oxidation flux in basophil.

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Materials and methods

Basic information on basophil membrane

Basically, the basophil membrane has a thickness about 5 nm. The basophile membrane can be prepared according to the method described by Lorenz et al (2003). In this study, this information was used for further simulation test.

Simulation test to determine the oxidation flux change

The simulation test to determine the oxidation flux change based on nanomedicine technique is used. The technique namely "process: oxidation flux" was used. Briefly, this simulation technique integrates both the classic Deal-Grove's model and Massoud's model, which both describe the oxidation growth process (Deal and Grove 1965). Specifically, this technique investigates the effect of different parameters and conditions on oxidation process by looking into the oxidation flux (Deal and Grove 1965). It gives users the freedom to adjust critical parameters and conditions in the process, such as oxidant condition, time, initial oxide thickness, temperature, pressure, crystal orientation, as well as an opportunity to choose between the Deal-Grove's or Massoud's model, or a combination of both (Deal and Grove 1965). The primary condition in this study is wet condition, temperature = 37 degree Celcius, operated time = 1 minute and oxygen pressure = 0.1 atmosphere according to the normal situation in blood stream. Variation of transmembrane ion concentration difference is used as simulating condition. Also, the control system with no oxidation basophile membrane is studied.

Result

According to the simulation test, the transmembrane oxidant concentration is steadily equal to 3e + 19/cm^3 at any transmembrane ion concentration difference (thickness) (Figure 1). For control data, no transmembrane oxidant concentration can be detected.

Discussion

The oxidation process is one of the most important natural processes. It is implemented in processes such as the gate dielectric growth. Oxidative change in hypersensitivity is believed to be an important process in the pathogenesis. Contrary to such a classical view, accumulating evidence indicates that upon stimulation of divergent receptor systems, reactive oxygen species (ROS) are intentionally produced and even required for appropriate signal transduction and biological responses (Suzuki et al 2005). More recent research reveals that ROS may also play an important role in mast cell activation by divergent allergy-relevant environmental substances, for instance heavy metals and polycyclic aromatic hydrocarbons (Suzuki et al 2005).

The change in transmembrane oxidative flux is believed to be a possible mechanism underlying basophil stimulation. In this work, the author uses the new technique in nanomedicine to determine the oxidation flux in the basophil. Of interest, no change of flux can be detected. This result is concordant with the report of (Lippert et al 2000) that antihistamines had no effect on calcium flux in resting or stimulated cells. In addition, it also agrees with the findings of Lindau and Fernandez (1986) that antigenic stimulation did not cause significant changes in the ionic conductances in a patch-clamp study of histamine-secreting cells, which suggests that these cells use a mechanism different from ionic currents in stimulus-secretion coupling. Indeed, Tedeschi et al (1989) proposed that there being receptor-operated, but not voltage-operated, calcium channels in the basophil leucocyte plasma membrane. Therefore, this work can support the

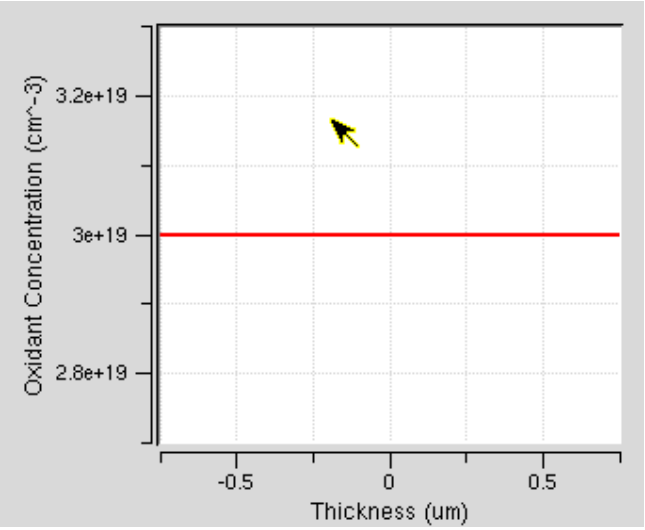

Figure 1 Oxidation flux of basophil. The transmembrane oxidant concentration is steadily equal to 3e + 19/cm^3 at any transmembrane ion concentration difference (thickness).

finding that the oxidation flux change is not an important part in the pathogenesis of basophil-related hypersensitivity.

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