Effect of Middle-Wave Ultraviolet Irradiation and Red Light on Degranulation of Peritoneal Mast Cell in Rats

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Middle-wave UV-irradiation inhibits liberator-induced histamine release from mast cells. Red light stimulated liberator-induced degranulation of mast cell. The existence of a membrane-dependent system activated by long-wave (red) light in mammalian cells is discussed.

Key Words: *UV-irradiation; red light; mast cells; histamine*

UV-irradiation evokes a variety of biological reactions, in particular histamine release from mast cells (MC). MC play an important role in various pathological processes such as anaphylactic shock, inflammation, malignant growth, and infection [1]. Noncytotoxic degranulation of MC and histamine release are realized via exocytosis, where the key role is played by Ca^{2+} [10]. MC exocytosis induced by liberators (substance 48/80, calcium ionophore A23187) is a good experimental model for studying modifications of membrane-bound reactions to different factors not associated with cytotoxic effects. This study investigated the effect of UV-irradiation on rat MC and the possibility of its modification.

MATERIALS AND METHODS

Purified fraction of peritoneal MC from female Wistar rats was used to study membrane modifications induced by UV-irradiation. The rats were decapitated under chloroform narcosis. MC were isolated as described previously [1]. The purity of MC fraction was 90-95%.

Immediately after the isolation MC were resuspended in a medium and irradiated with UV and/or a monochromatic red light (RL). The source of middle-wave UV-radiation (290-320 nm, UVB) was a high-pressure mercury lamp (1000 W). This range was chosen because recordable changes in MC membranes are caused only by UVB-radiation [4,5,7]. The UVB band (303-310 nm) was isolated with UFS-2 and ZhZ-3 filters. Secondary UVB radiation (below 300 nm) was cut off with a BS-5 glass filter. UVB-radiation was applied in doses of 3.9 and 6.0 kJ/m^2 at the intensity of 15 W/m^2. RL-radiation (680 nm) was applied in a dose of 120 J/m^2, which provides maximum recovery according to our previous data on cell culture. Monochromatic RL was obtained from a DRSh-1000 lamp using a diffraction spectrograph with a 1 nm/mm linear dispersion (light intensity at 680 nm was 0.2 W/m^2). In some experiments MC were irradiated with white light in doses of 11.7, 19.5, and 39 kJ/m^2. After irradiation, substance 48/80, a selective histamine liberator (25-1 μg/ml), was added to MC for 10 min at 37°C. Histamine was determined by a fluorescent technique [7] using a Hitachi-850 spectrofluorimeter. The percent of released histamine was calculated by the difference between sediment and supernatant fluorescence.

RESULTS

UVB-radiation in a dose of 0.5-9.0 kJ/m^2 did not induce MC degranulation. Histamine release from non-irradiated MC in the presence of substance 48/80 in doses of 0.25 and 0.5 μg/ml was 17.27 and 20.79%,

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respectively. UVB irradiation (3.9 kJ/m^2) decreased the release of histamine (Fig. 1). It can be concluded that UVB-radiation modifies MC response to histamine liberator. These results agree with published data showing that UVB-irradiation of rat peritoneal MC in doses below 7.2 kJ/m^2 inhibited substance 48/80-induced histamine release without causing MC degranulation [5].

Combined irradiation with UVB and RL had the same effect on a liberator-induced 48/80 histamine release as UVB alone, *i. e.* reduced the percent of released histamine compared to the control. Thus, long-wave radiation did not reverse the membrane modifications induced by UVB. Similar principally new photoprotective system activated by optical stimuli (most efficiently by 680-nm radiation) was described in yeast cells [2]. We expected similar photoprotective effect in our model of UV-induced impairment of membrane-dependent function of MC. However, in our study RL applied in doses stimulating photoprotection under conditions of photorestoration and photoprotection from reproductive death did not restore histamine release from MC suppressed by UVB-irradiation.

At the same time, RL stimulated liberator-induced histamine release by 50% compared to nonirradiated MC (Fig. 1). Thus, RL in test doses modified the processes induced by selective 48/80 liberator.

According to published data, monochromatic light simulates the division of HeLa cells and DNA and RNA synthesis in yeast and bacteria with a clear-cut maximum at 680 nm [6]. In our experiments, peritoneal MC were irradiated with a monochromatic light at 405, 436, 500, 578, 630, and 680 nm and then incubated with 48/80 liberator in optimum concentration (0.5 μg/ml). The most pronounced peak of histamine release (50% above the control level) was observed at 680 nm, 405 nm produced only a minor increase (8%), while other wavelengths had no effects (Fig. 2). The light-induced modification of histamine release from MC is not associated with photodynamic effects, since our experiments with white light (11.7-39 kJ/m^2) revealed no effects on both spontaneous and 48/80 liberator-induced histamine release.

Our data suggest that mammal cells possess a membrane-dependent system which is activated by long-wave RL. It can be assumed that MC membranes have photoactive RL receptors, but no compounds with absorption in the far red band of the spectrum were isolated until now. It was hypothesized that this light can be accepted by terminal cytochrome oxidase of the respiratory chain (presumably, an intermediate, partly oxidized, partly reduced form of the enzyme) [6]. Changes in the redox state can modify many parameters of cell homeostasis. The nature of a long-wave RL acceptor mediating the described effect remains to be clarified in further investigations.

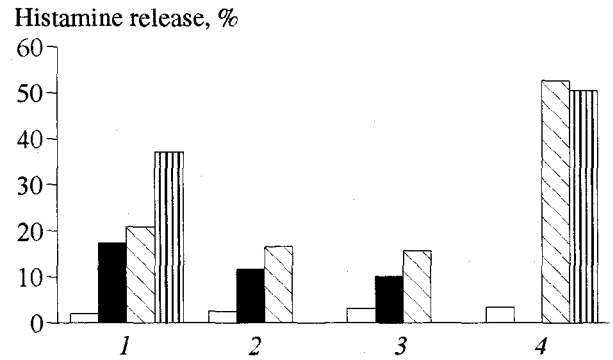

Fig. 1. Effect of UVB and red light (680 nm) on spontaneous (open bars) and stimulated release of histamine from rat peritoneal mast cells induced by substance 48/80 in concentrations of 0.25 (filled bars), 0.5 (oblique shading), and 1 (vertical shading) μg/ml. *1)* control; *2)* UVB-irradiation; *3)* UVB-irradiation+red light; *4)* red light.

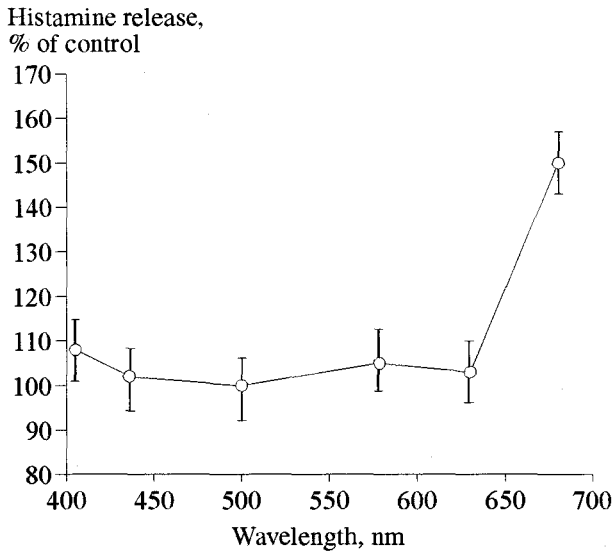

Fig. 2. Photostimulation of histamine release from rat peritoneal mast cell.

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