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제 목	Inhibitory effects of Cortex Mori on Compound 48/80-induced Anaphylactic Shock and Cutaneous Reaction
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Although active systemic anaphylaxis and passive cutaneous anaphylaxis have been empolyed to study anaphylactic hypersensitivity, it is difficult and time-consuming to induce these reactions in experimental animals. In recent, Jun et al have found a simple method to induced anaphylactic hypersensitivity such as anaphylactic shock(AS) and cutaneous reaction(CR) using compound48/80. Cortex mori (*Morus alba* L.), the root bark of mulberry tree has been used as an antiphlogistic, diuretic, and expectorant in herbal medicine. The purpose of this study was to determine whether the methanol extract of Cortex mori could inhibit the compound 48/80-induced AS and CR. To induce AS, various doses of compound 48/80 (5, 7.5, 10, 15 ug/gm B.W.) were injected intraperitoneally (i.p.) into ICR mice. The animals were pretreated by three injection(i.p.) of Cortex mori before compound 48/80 administration. Peripheral blood was collected from the right ventricle to estimate the level of serum histamine at 15 minutes after the injection (i.p.) of various concentration of compound48/80. Mortility rate, mean death time and mesenteric mast cell degranulation rate were evaluated over a 72 hour period. To estimate the effect of Cortex mori on compound 48/80-induced cutaneous reaction, various doses of compound 48/80 with or without Cortex mori were injected intradermally(i.d.) into the shaved flank of Sprague-Dawley rats, and the blue cutaneous patchs induced by Evans' blue injection at the compound 48/80 alone and Cortex mori plus compound 48/80 injection sites were observed. As a parameter of these reactions, the levels of histamine in the supernatant, calcium uptake and intracellular cAMP of RPMC were measured. Results are 1)compound 48/80-induced mortility rate, mean death time, mesenteric mast cell degranulation rate, and serum histamine level in ICR mice were significantly inhibited by pretreatment of Cortex mori, 2) cutaneous reaction inducd by compound48/80 was well developed in Sprague-Dawley rat, but Cortex mori inhibited the compound 48/80-induced blue patch formation remarkably, 3) the compound 48/80-induced degranulation, histamine release and calcium uptake of RPMC pretreated with Cortex mori were significantly inhibited, compared to those of control without Cortex mori pretreatment, and 4)the level of cAMP of RPMC was reduced by the increased concentration of compound 48/80, pretreatment of Cortex mori not only inhibited the compound 48/80-induced reduction of cAMP but also significantly increased the level of cAMP naturally. From the above results, it is suggested that Cortex mori has an some substances with an ability to inhibits the compound 48/80-induced AS,CR, and mast cell activation.