Th2 dominance within Th1/Th2 balance in the acquired arm has been thought to play a central role in the induction of allergic sensitization as well as in the chronic inflammatory cascades of various allergic diseases. It has recently been proposed that two major distinct subsets of dendritic cells (DCs) in the innate arm are arranged to regulate the immune responses in vivo: DEC-205+ DCs having the capacity to establish Th1 polarization while 33D1+ DCs to establish Th2 dominance. We have previously reported that 33D1+ DCs could be successfully depleted from mice with anti-33D1-specific monoclonal antibody (mAb) treatment and that IL-12 secretion was elicited when the 33D1+ DC-depleted mice were stimulated with LPS (Cancer Immunol. Immunother., 59: 1083-1095, 2010). Moreover, we have recently demonstrated that the fetal loss was induced by the depletion of 33D1+ DCs during perinatal period mediated through transient IL-12 secretion by Th1 up-regulation (Immunobiol., 217: 951-961, 2012). Based on these findings, we examined the effect of 33D1+ DC-depletion on allergic airway sensitization against ovalbumin (OVA) in mouse model in vivo. In which, mice were sensitized intraperitoneally with OVA plus alum, and were challenged intranasally with OVA solution. As expected, when 33D1+ DCs were previously depleted before the sensitization, anti-OVA IgE level in the serum and various allergic symptoms such as sneezing or nasal scrubbing times were apparently decreased as compared with control untreated mice. These results suggest that either depletion/inhibition of innate 33D1+ DCs or activation of DEC-205+ DCs may offer a new therapeutic strategies for various allergic diseases by altering Th1/Th2 balance in vivo.