Respiratory viruses are responsible for tissue damages and local inflammation. The best strategy to control their severity is to limit the infection while maintaining an efficient immune response. Given this context, the case when the macrophage is the target cell of infection is of interest. Indeed, pulmonary macrophages (i) are responsible for inflammation and viral destruction by phagocytosis and (ii) participate in the induction and orientation of the adaptive immune response. Consequently, macrophage infection hampers the whole immune response. The interaction between macrophages and virus during the first steps of infection has not been throughly investigated in experimentale studies and is not detailed in models of immune response. Consequently, the influence of macrophage – virus interactions on the infection resolution is unknown. Here, we propose an original model of the immune response centred on the macrophage – virus interactions. We represent all macrophage infectious statuses, their immune functions, and the interactions between innate and adaptive responses taking into account the cytokines regulations. We use the model to study the relative influence of macrophage – virus interactions on the infection resolution by a multivatiate sensitivity analysis. Then, we explore the influence of macrophage immune functions by considering two levels of host susceptibility and viral virulence. We conclude that both repilication rate of the virus and host capacity to synthetize anti-viral cytokines are key for infection resolution.