Abstract:

Diesel exhaust particles (DEP) were described as potent adjuvant in the induction and maintenance of allergic diseases, suggesting that they might play a role in the increase of allergic diseases in the industrialized countries. However, the cellular basis by which these particles enhance allergic immune responses is still a matter of debate. Thus, we exposed immature murine bone marrow-derived dendritic cells (BMDC) to different particles or particle-associated organic compounds in the absence or presence of the maturation stimuli lipopolysaccharide (LPS) and analyzed the cellular maturation, viability, and cytokine production. Furthermore, we monitored the functionality of particle-exposed BMDC to suppress B cell isotype switching to immunoglobulin (Ig) E. Only highly polluted DEP (standard reference material 1650a [SRM1650a]) but not particle-associated organic compounds or less polluted DEP from modern diesel engines were able to modulate the dendritic cell phenotype. SRM1650a particles significantly suppressed LPS-induced IL-12p70 production in murine BMDC, whereas cell-surface marker expression was not altered. Furthermore, SRM1650a-exposed immature BMDC lost the ability to suppress IgE isotype switch in B cells. This study revealed that highly polluted DEP not only
interfere with dendritic cell maturation but also additionally with dendritic cell function, thus suggesting a role in T(h)2 immune deviation.