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Aryl hydrocarbon receptor regulates cockroach allergen induced lung inflammation through controlling the recruitment and function of mesenchymal stem cells

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Exposure to cockroach allergen can lead to allergic sensitization and an increased risk of developing asthma. Recent studies have suggested that aryl hydrocarbon receptor (AhR) can sense not only environmental pollutants but also microbial insults. To test whether AhR can sense allergens and modulate allergic responses, we examined cockroach allergen induce AhR activation in mesenchymal stem cells (MSCs) and lung inflammation in mouse model of asthma with wild-type (WT) and AhR-deficient (AhR-/-) mice. AhR mediated MSC migration was investigated using Transwell migration assay and GFP+MSCs administration in mouse model. The role of migrated MSCs in suppressing lung inflammation and macrophage polarization was further investigated. Our studies demonstrated that AhR signaling was activated with increased expression of cyp1a1 and cyp1b1, downstream genes of AhR when MSCs were exposed to cockroach allergens. Compared to WT mice, cockroach allergen treated AhR-/- mice showed exacerbation of lung inflammation. AhR mediated allergen-induced inflammation was further validated by using the AhR agonist, 2, 3, 7, 8-Tetrachlorodibenzo-p-dioxin (TCDD). Moreover, TCDD promoted CRE-induced MSCs migration while the AhR antagonist CH122319 suppresses MSCs migration in our Transwell assays. Furthermore, CRE-challenged AhR-/- mice displayed less migrated MSCs to the lungs compared to WT when GFP+MSCs were administrated intravenously. Additionally, the administration of MSCs significantly attenuated allergic inflammation which was in part rescued by TGFβ1 neutralizing antibody. Interestingly, macrophages from MSCs treated mice exhibited M2 phenotypes with increased expression of Agr-1, FIZZ-1 and Ym-1. The macrophage polarization was possibly induced by MSCs and confirmed by co-culturing MSCs with bone marrow-derived macrophages. Our findings provided evidence for a previously unidentified pathophysiological function of the AhR and suggested that AhR may be critical in modulating environmental allergen induced immune inflammation.

Biography

Peisong Gao is currently Associate Professor at The Johns Hopkins University School of Medicine in Baltimore, Maryland. He received his MD degree and Pulmonary Medicine Training in The Fourth Military Medical University, China. From July 1997 to January 1999, he was a Visiting Research Fellow in Oxford University. He subsequently moved to the University of Wales Swansea pursuing a PhD working in Molecular Genetics of Asthma. In 2002 he became a Postdoctoral Fellow in the Division of Allergy & Clinical Immunology at Johns Hopkins. In 2008, he was promoted to Assistant Professor. His research has been greatly recognized by several awards including the 2004 Research Excellence Award, the 2007 Interest Section Award and Outstanding Pediatric Allergy, Asthma and Immunology Award from AAAAI. His studies mainly focus on gene, environment and development of asthma. He has published more than 60 papers in reputed journals.

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