Aromatic hydrocarbon receptor provides a link between smoking and rheumatoid arthritis in peripheral blood mononuclear cells: a commentary

Sirs,

We have read with great interest the article “Aromatic hydrocarbon receptor provides a link between smoking and rheumatoid arthritis in peripheral blood mononuclear cells” by Qian et al. (1). The article focused on the potential relationship between smoking, aromatic hydrocarbon receptor (AHR), and rheumatoid arthritis (RA). It was worthy of recognition that the article linked the basics to the clinic, providing valuable knowledge for us to further understand the pathogenesis of environmental factors such as smoking on RA, and further deepening our understanding of the aetiology of RA. However, caution must be exercised when assessing their data and conclusions. Several limitations of the article design and analysis were listed below, which require further analysis and discussion.

Firstly, reverse transcription-polymerase chain reaction could only reflect the expression level of AHR repressor, and cytochrome P4501A1 at the messenger RNA level, and western blot should be used to further elucidate the expression levels of these genes from the protein level (2, 3). In addition, flow cytometry could be used to detect the ratio of AHR-positive peripheral blood mononuclear cells (PBMCs) in peripheral blood to total PBMCs, and to better elucidate the role of AHR in the pathogenesis of RA. It could not only enrich experimental data, but also provide provided stronger support for the establishment of conclusions.

Secondly, in the Materials and Methods section, the author also mentioned the detection of erythrocyte sedimentation rate, C-reactive protein and anti-cyclic citrullinated peptide anti-CCP antibodies of all RA patients. These indicators are commonly used as clinical indicators in the diagnosis of RA and evaluation of disease activity (4). The results of these indicators should be listed in the results section, combined with experimental data to further explore the relationship between AHR and RA disease activity.

Thirdly, RA is a chronic autoimmune disease in which multiple inflammatory factors are involved in the development of RA. Detection of inflammatory factors closely related to RA such as IL6, should be added to the article (5). The expression level of inflammatory factors was detected to reflect whether smoking could aggravate the inflammatory response of RA, and thus accelerate the progression of the disease, more fully interpret the relationship between smoking, AHR and RA.

In conclusion, the article provided new ideas for RA research. AHR might be an important bridge for environmental factors involved in the pathogenesis of RA, and might also be a potential target for the diagnosis of RA. Of course, to further study the complex relationship between these indicators, it is still necessary to conduct research in a larger sample.

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References


