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Indoleamine 2,3-dioxygenase decreases humoral alloimmunity in primary human peripheral blood mononuclear cells by pathways different than those implicated in its effect on T-cells

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Background: Chronic antibody-mediated rejection remains a major cause of late graft loss. As regards to cellular alloimmunity, the immunosuppressive properties of indoleamine 2,3-dioxygenase (IDO) are well-defined, but less are known about its effect on humoral alloimmunity.

Methods: For this purpose, we developed a method for the induction of humoral alloimmunity in one-way mixed lymphocyte reaction (MLR) and we measured it with an antibody-mediated complement-dependent cytotoxicity assay against resting cells like the stimulator cells of the above MLR. In parallel cellular alloimmunity was assessed in two-way MLRs. The IDO inhibitor 1-methyl-DL-tryptophan was used for evaluating the role of IDO. In order to investigate if the pathways known to play role in the effect of IDO on T-cells are applied in humoral alloimmunity, the general control nonderepressible-2 (GCN-2) kinase activator tryptophanol and the aryl hydrocarbon receptor (AhR) inhibitor CH223191 were used.

Results: Regarding cellular autoimmunity, the IDO inhibitor increased it, whereas the GCN-2 kinase activator decreased it. Unexpectedly, the AhR inhibitor decreased cellular alloimmunity. Concerning humoral alloimmunity, our results demonstrated that IDO decreases it in a GCN-2 kinase and possibly in an AhR independent way.

Conclusion: IDO decreases humoral alloimmunity in primary human PBMCs by pathways different than those implicated in its effect on T-cells

Biography

Maria Sounidaki received her Bachelor degree in Molecular Biology at Aristotle University of Thessaloniki, Greece, and Master degree in Biomedical Sciences from University of Maastricht, Netherland. She is currently pursuing her PhD in Department of Nephrology, Faculty of Medicine, University of Thessaly in the field of Immunology and Cellular Metabolism (especially within the context of chronic kidney disease). She is a Pre-doctoral Research Fellow at the Clinic of Nephrology and Hypertension, Diabetes and Endocrinology at the Otto von Guericke University Magdeburg in Germany where she is performing *in vivo* experimental studies in mouse models of Interstitial nephritis.

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