**INTRODUCTION AND AIMS:** Despite therapeutic immunosuppression, a significant proportion of transplanted patients develop donor-specific antibodies (DSA), which are currently recognized as the first cause of allograft failure. Generation of antibodies against protein antigens (including donor HLA) results from a thymus-dependent (TD) humoral response, which means that B cells need to receive a co-stimulation signal from activated follicular helper T cells (Tfh) to differentiate into plasmacells.

**METHODS:** In this study, we test whether profiling of circulating Tfh (cTfh) could predict the ability to mount a TD humoral response in 36 renal transplanted patients and 9 healthy controls. We took advantage of the 2015 influenza vaccination campaign, which provided a normalized setting of antigenic stimulation. The number of cTfh, their polarization profile, and ability to up-regulate i) helper molecules (CD40L and ICOS) and ii) the activation marker CD25 following in vitro stimulation in presence of patients' own plasma (with IS drugs) were measured prior vaccination. These parameters were then compared between responders and non-responders to influenza vaccine.

**RESULTS:** While most of the characteristics of cTfh profile were similar between the two groups, we observed that responders showed a significantly higher proportion of cTfh17 that upregulated CD25 expression after in vitro stimulation. We performed a posteriori analysis of the cTfh profile of 15 transplanted patients at the time of DSA appearance and found that the proportion of cTfh17 cells that upregulated CD25 after in vitro stimulation was similar to responder to influenza vaccine.

**CONCLUSIONS:** We concluded that the ability of the cTfh17 subset to be activated in vitro predicts TD antibody response and might be used as non-invasive biomarker to identify transplanted patients at risk to develop DSA.

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**FP697 ACTIVITY OF CIRCULATING Tfh17 PREDICTS HUMORAL RESPONSE TO THYMUS-DEPENDENT ANTIGENS**

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**METHODS:** We performed a meta-analysis and the review of literature revealed five and seven additional candidate microRNAs of AR, respectively. Quantitative real-time polymerase chain reaction confirmed that a total of 5 microRNAs (hsa-miR-4488, hsa-miR-4532, hsa-miR-30a-3p, hsa-miR-185-5p, hsa-miR-223-3p) maintained their differential expressions, and the combination of these 5 microRNAs could distinguish AR from stable graft function.

**RESULTS:** These preliminary results showed an overexpression of 5 microRNAs in patients with an acute or chronic rejection compared to stable graft function. These microRNAs include miR-142-3p, miR-106b-3p, miR-101-3p, miR-185-5p, and miR-21-5p. These microRNAs are useful to verify the presence of miRNAs in renal tissue and verify their expression also after transplantation. For this reason it would be useful to prevent kidney rejection has grown up. The study could be a new approach to prevent kidney rejection.

**CONCLUSIONS:** This study demonstrated that the profiles of urinary exosomal microRNAs from 108 kidney transplant recipients were extracted. The candidate microRNAs for the diagnosis of AR were selected based on literature. The levels of selected candidate microRNAs were further confirmed by quantitative real-time polymerase chain reaction. The levels of these microRNAs were significantly altered in patients with AR compared to those with stable graft function. The levels of urinary exosomal microRNAs could be potential biomarkers for the early diagnosis of AR.