Infinite Science Publishing

β1-adrenergic receptor autoantibody levels are higher in patients with Graves' hyperthyroidism than in matched healthy controls and decrease after anti-thyroid treatment

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Background: Graves' disease (GD) is driven by autoantibodies targeting the thyroid-stimulating hormone receptor which belongs to the G-protein-coupled receptor (GPCR) superfamily. These autoantibodies (TRAb) result in hyperthyroidism, which entails symptoms from many organs, including the heart with risk for tachyarrhythmia, atrial fibrillation and heart failure. In previous studies of GD patients with heart complications, antibodies targeting cardiovascular GPCRs including the β1-adrenergic receptor (B1R) were shown to be frequent. However, most of these studies were relatively small and used non-standardised peptide based ELISA methods or complex cell assays for antibody assessment. In this study, we used a standardised ELISA assay (CellTrend) to analyse antibodies directed against conformational B1R epitopes (B1RAb) to investigate if such antibodies are increased in GD patients compared to healthy controls, are influenced by antithyroid treatment and correlate with TRAb levels or heart related symptoms.

Methods: This is a preliminary report of results from GD patients in an ongoing longitudinal observational prospective study. Premenopausal women in profound hyperthyroidism were included and symptom scores, TRAb and B1RAb were measured at baseline and after 7.5 months of treatment. We here present results from the first enrolled 43 patients and matched controls.

Results: B1RAb levels were higher in patients at baseline compared to healthy controls (median 1.9 vs 0.9 µg/ml, P<0.0001). B1RAb levels decreased after 7.5 months of treatment (median 1.6 µg/ml, P=0.001), but remained higher than in controls (P=0.0008). B1RAb levels did not correlate with TRAb levels or heart related clinical symptoms.

Conclusion: B1R autoantibodies were increased in GD patients at study inclusion and decreased with treatment. Continued studies include analysis of antibodies against other GPCRs expressed in the cardiovascular system and the relation to biomarkers of heart function.