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Summary of Research to Date
April 2023

"STAT3-mediated regulation of the human immune response-unraveling the complexities of disease pathogenesis of Job Syndrome".

Job syndrome is caused by defects in a gene called STAT3 – this is a critical gene that co-ordinates the function of many systems including the immune system and connective tissue/musculoskeletal systems. STAT3 functions to regulate the expression of many other genes in cells throughout the body. Due to errors in the STAT3 gene, various clinical symptoms can develop – particularly recurrent infections of the skin and lung, and atopy/allergy. We have been very interested in understanding exactly how these errors in the STAT3 gene cause these clinical problems. To address this we have been interrogating the behavior of individual immune cells (B and T cells) and how this differs from corresponding B and T cells in healthy individuals. We have been specifically intrigued by how altered errors in STAT3 alters it ability to control expression of many of its target genes. The data from these studies identified particular defects in the ability of these immune cells from patients with Job syndrome to carry out their specialized functions to protect against infection and prevent the development of atopic and allergic conditions. We also found key genes that are not expressed appropriately due to poor function of STAT3 – these findings us to investigate conditions very similar to Job syndrome in terms of genetic causes to better understand how these diseases develop and how they could potentially be better treated.