Re-visiting the HLA dogma

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Unravelling the intricacies of the human leukocyte antigen (HLA) system has been fundamental to our understanding of the immunobiological discrimination of self from non-self. In the context of solid organ transplantation, the allo-immune response is predominantly driven by a varying but inevitable degree of HLA mismatch (classically HLA class I and II molecules) between donor organs and the recipient host. In general, donor–recipient HLA mismatching tends to be maximal for solid organs where prospective HLA matching is not possible due to logistic reasons. This is true for lung transplantation, although rapid screening cellular and antigenic assays at the time of transplantation and thereafter can help detect, mitigate and manage the risks associated with donor-specific antibody production in the recipient. More recently, the newly developed epitope analysis is a significant step forward for ever finer precision deciphering of immunogenic mismatch-driven donor-specific HLA antibody production (DSA) [1] and graft and recipient outcomes [2].