HIV-SPECIFIC T CELL CLONOTYPES MAY CONTRIBUTE TO THE ABACAVIR DRUG HYPERSENSITIVITY REACTION VIA HETEROLOGOUS IMMUNITY

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Drug hypersensitivity (DHR) is a common immune mediated reaction which can be associated with severe illness including rash, hepatitis, DRESS/SJS and occasionally death. How sensitization occurs and the underlying immunological mechanism by which a drug can induce an immune response is unclear. One proposed theory is that of heterologous immunity whereby virus-specific T cells may cross-react against drug altered peptide repertoire presented on autologous HLA molecules. We provide proof-of-principle *in vitro* evidence that pre-existing virus-specific memory T cell clonotypes can recognise drug altered peptide repertoire presented on autologous HLA. Here, we show that that a human HIV Gag TW10/HLA-B57-specific CD8 memory T cell clone recognizes autologous HLA-B57, but only in the presence of abacavir. Results presented here are the first to suggest that HIV-specific memory T cells may themselves participate in abacavir induced HSR via heterologous immunity.