Title:	TLR9 signalling activation via direct ligation and its functional consequences in CD4 + T cells
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Abstract:	CpG Oligodeoxynucleotides (ODNs) are established TLR9 ligands; however, their functional responses in CD4+ T cells are believed to be independent of TLR9 and MyD88. We studied ligand-receptor interactions of ODN 2216 and TLR9 in human CD4+ T cells and assessed their consequences in terms of TLR9 signalling and cell phenotype. We demonstrated that the uptake of ODN 2216, a synthetic TLR9 agonist, is controlled by TLR9 signalling molecules and results in an increase in the expression of TLR9 signalling molecules, regulated via a feedback mechanism. Next, the uptake of ODN 2216 resulted in TLR9 signalling dependent but MyD88 independent increase in expression of TGF-β. Finally, ODN 2216 treated CD4+ T cells showed an anti-inflammatory phenotype that was similar to Th3 type of regulatory T cells. These Th3-like cells were able to suppress the proliferation of untreated CD4+ T cells. Collectively, our results demonstrate a direct and interdependent relationship between ODN 2216 uptake and TLR9 signalling in CD4+ T cells. Our findings thus pave the way for future research to explore direct modulation of adaptive immune cells, using innate immune ligands, to subvert exaggerated inflammatory responses.
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