DOCK2 couples with LEF-1 to regulate B cell metabolism and memory

response	
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Dedicator of cytokinesis 2 (DOCK2) is essential for the B cell differentiation, BCR signaling and humoral immune response. However, the role of DOCK2 in the memory response of B cell is unknown. By using two DOCK2 deficient patients, we found that the memory B cells were decreased and the early activation of DOCK2 deficient memory B cells was abolished to the degree of naïve B cells due to the decreased expression of CD19 and CD21 mechanistically. Interestingly the expression of LEF-1, a negative regulator of CD21, was increased in DOCK2 deficient B cells. This was linked to the increased expression of HIF-1? and cell metabolism, which in turn affected the ER structure. Finally, the reduction of memory B cells in DOCK2 patients was due to the

increased apoptosis, which might be related with the increased metabolism. $\ensuremath{\textcircled{\text{\scriptsize C}}}$ 2020

B cell
DOCK2
LEF-1
B lymphocyte receptor
CD19 antigen
complement component C3d receptor
dedicator of cytokinesis 2
hypoxia inducible factor 1alpha
lymphoid enhancer factor 1
Rac protein
unclassified drug
animal cell
Article
B lymphocyte activation
B lymphocyte differentiation
cell metabolism
clinical article
controlled study
female
human
human cell
male
memory cell
mouse
nonhuman

priority journal