Supplemental Methods

Generation of LZRS-FOXP1-IRES-YFP

pcDNA3.1-FOXP1-myc-his encoding human FOXP1 was obtained from Daniel Simon (Harvard medical school, Boston, MA). The myc- and his-tag at the 3’end of the coding region were replaced by a stop codon. FOXP1 was subsequently subcloned into LZRS-IRES-YFP via pcDNA4-TO to generate LZRS-FOXP1-IRES-YFP (referred to as FOXP1-IRES-YFP).

B-cell isolation

Buffy coats were obtained from Sanquin bloodbank (Amsterdam, the Netherlands). PBMCs were isolated by Ficoll separation. B-cells were subsequently obtained by MACS separation with a memory B-cell isolation kit (Miltenyi Biotech, Bergisch gladbach, Germany), or occasionally by FACSAria sorting of the CD19^+CD27^+ population using an APC-conjugated antibody against CD19 (BD Biosciences, San Jose, Ca) and a PE-conjugated antibody against CD27 (BD).

B cell cultures, retroviral transductions and siRNA-mediated knockdown

Isolated human B cells were cultured on CD40L-L cells\textsuperscript{2} IL-21(25 ng/ml, R&D systems, Abingdon, UK) and IL-2 (40U/ml, prospec, East Brunswick, NJ) in Iscove’s modifies Dulbecco’s medium (IMDM) containing 10% FCS, 100 units per ml of penicillin, and 100 μg per ml of glutamine (IMDM 10% FCS medium). For transduction, after culturing the cells for a minimum of 36 hours, cells were transferred to retronectin-coated plates and incubated with virus for 6-8 hours in IMDM 5% FCS medium. Thereafter, cells were cultured with CD40L-L cells, IL-21 and IL-2 in 10% FCS medium for 72 hours after which they were passaged and cultured either under
the same conditions or without CD40L-L cells. For microarray analysis, after transduction cells were cultured without cytokines for 3 days.

OCI-Ly3 and OCI-Ly10 were cultured in IMDM 20% medium, supplemented with 35 µM β-mercaptoethanol. OCI-Ly1, OCI-Ly7, and SUDHL6 were cultured in IMDM 10% FCS medium, U2932 was cultured in RPMI 10% FCS medium. For transductions, cell lines were transferred to retronectin-coated plates and incubated with virus for 24 hours. For siRNA-mediated knockdown, cells were transiently transfected using the Lonza nucleofection system. 5 ×10^6 cells were resuspended in Nucleofector kit T (Lonza, Basel, Switzerland) mixed with 2.5 µg siRNA against FOXP1 or sigenome non-targeting siRNA pool #2 (Dharmacon, Thermo scientific, Waltham, MA) and pulsed with program G16. FOXP1 expression levels were determined by immunoblotting with anti-FOXP1 antibody (ab16645) from Abcam. BCL6 expression levels were determined by immunoblotting with anti-BCL6 antibody (ab16645) from DAKO.

RT-qPCR

Total RNA was isolated using the Rneasy micro kit (Qiagen, Valencia, CA) or Trizol (Invitrogen), followed by isopropanol precipitation, and reverse-transcribed into cDNA with first strand buffer (Invitrogen), Moloney murine leukemia virus reverse transcriptase (Invitrogen, Carlsbad, CA), dNTPs, and Oligo (dT). For quantitative polymerase chain reaction (PCR), we used a Roche LightCycler 480 and mixed sensimix (Bioline, London, UK) with cDNA and primer pairs for the indicated genes and normalized using primer pairs for HPRT. Primer sequences can be found in supplemental table II.
**Microarray analysis**

Biotinylated complementary RNA was amplified with a double in vitro transcription, according to the Affymetrix small sample labeling protocol vII (Affymetrix, Santa Clara, CA, USA). The biotinylated complementary RNA was fragmented and hybridized to the HG-U133 Plus 2.0 GeneChip oligonucleotide arrays according to the manufacturer’s instructions (Affymetrix). Fluorescence intensities were quantified and analyzed using the GCOS software (Affymetrix). The threshold for significant change was set to a P-value of $\leq 0.0025$. Genes with 100% absent call were considered not to be biologically relevant and were removed. Genes that showed significant differential expression upon FOXP1 overexpression and/or knockdown in at least two distinct experiments were considered to be regulated by FOXP1. The microarray data is deposited in GEO (accession number GSE51382, http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?token=ijktmuaytlevbav&acc=GSE51382). Data were analyzed and heatmaps were generated using R2: microarray analysis and visualization platform (http://r2.amc.nl). The gene ontology (GO) biological pathways were identified using DAVID software (Database for Annotations, Visualization and Integrated Discovery)\textsuperscript{3,4}

**Chromatin immunoprecipitation (ChIP) and sequencing**

ChIP-seq was performed using 5 µg of anti-FOXP1 (ab16645) from Abcam. De novo motif discovery was performed using CisModule function incorporated in Cisgenome software.\textsuperscript{5} Similarity search with known transcription factor binding motifs was performed using TOMTOM motif comparison tool.\textsuperscript{6} Primers used for ChIP-qPCR are listed in supplemental table III.

**Flow cytometry**
Propidium iodide, eFluor 670, CFSE, GFP and/or YFP fluorescence were acquired on a FACSCanto (BD Bioscience) flowcytometer and analyzed using FlowJo software (TreeStar, Ashland, OR).

**Caspase-Glo 3/7 assay**

Activation of Casase 3 and/or Caspase 7 was measured using the Caspase-Glo 3/7 assay (Promega, Madison, WI) according to the manufacturer’s instructions. Luminescence was measured in a Glomax microplate-reading luminometer (Promega). Live cell numbers were determined by flow cytometry and luminescence values were normalized for numbers of live cells in each sample.

**Proliferation and cell cycle analysis**

For eFluor 670 or CFSE labeling, cells were washed in PBS and resuspended in 100 µl PBS. An equal volume of 2 µM eFluor 670 (eBioscience, San Diego, CA) was added, and cells were incubated at 37°C for 15 min, quenched in 100% FCS, and washed twice with medium containing 10% FCS. For cell cycle analysis, cells were fixed with ethanol, washed in PBS 0.5% BSA and incubated for 20 minutes with 50 µg/ml RNAse A and 50 µg/ml Propidium Iodide (invitrogen, Carlsbad, CA) in PBS 0.5% BSA.

**Survival analysis of DLBCL patients**

To investigate if the 7 apoptotic gene panel could have prognostic clinical significance in DLBCL, we analyzed GEM data from 498 DLBCL patients treated with rituximab-CHOP therapy, by means of the R2 microarray analysis and visualization platform (http://r2.amc.nl).
The DLBCL patients were either stratified in two groups by k-means clustering of the 7 genes, or in a low or high expression group after ranking the patients according to the mean z-score or the summed rank of the 7 genes. Kaplan-Meier plots were generated for analysis of overall and progression-free survival.
Supplemental figures and tables

Supplemental figure 1. Kaplan-Meier plots of the overall survival (OS) or progression-free survival (PFS) of 498 DLBCL patients treated with rituximab-CHOP therapy

Patients were stratified in 2 groups by expression of the 7 FOXP1-repressed pro-apoptotic genes. (A) PFS of DLBCL patients stratified in two groups by k-means clustering of the 7 genes. The 5-year PFS is 64% for the patient group with overall higher (group 0; red) versus 45% in the group with overall lower (group 1; blue) expression of the 7 genes. (B) PFS of DLBCL patients stratified in two equal sized groups (separated at the median) by ranking the patients according to their mean z-score of the geneset. The 5-year PFS is 64% in the high (blue) versus 48% in the low (red) expressing group. (C) OS (left) and PFS (right) of DLBCL patients stratified in two equal sized groups (separated at the median) by ranking the patients according to the summed rank of the geneset. The 5-year OS is 72% in the high (blue) versus 49% in the low (red) expressing group, and the 5-year PFS is 64% in the high (blue) versus 47% in the low (red) expressing group. (D) OS of either ABC- (left) or GCB-type (right) DLBCL patients stratified in two groups by k-means clustering of the 7 genes. The 5-year OS of the ABC-type DLBCL patients (left) is 68% for the patient group with overall higher (group 0; red) versus 32% in the group with overall lower (group 1; blue) expression of the 7 genes. The 5-year OS of the GCB-type DLBCL patients (right) is 78% for the patient group with overall higher (group 0; red) versus 57% in the group with overall lower (group 1; blue) expression of the 7 genes. (E) (top) The color bar displays the z-scores (blue = low, red = high) of each of the 7 pro-apoptotic genes (from top to bottom): TP63, HRK, EAF2, TP53INP1, AIM2, RASSF6 and BIK. (middle) The heatmap displays the z-scores (green = low, red = high) of each of the 7 pro-apoptotic genes (from top to bottom): TP63, RASSF6, BIK, HRK, EAF2, TP53INP1 and AIM. The blue – red bar
displays the mean z-scores (blue is low; red is high) for the geneset, according to which the patients were ranked. (bottom) The heatmap displays the z-scores (green = low, red = high) of each of the 7 pro-apoptotic genes (from top to bottom): TP63, RASSF6, BIK, HRK, EAF2, TP53INP1 and AIM. The blue – red bar displays the mean z-scores (blue is low; red is high) for the geneset, and the rainbow-colored bar displays the summed ranks of the genes, according to which the patients were ranked.

Supplemental figure 2. FOXP1 ChIP-seq analysis: motif enrichment in FOXP1 binding regions and gene ontology analysis of FOXP1-bound genes.

(A) Distance of FOXP1 ChIP-seq peaks and randomized peaks from the TSS of the gene closest to the peak are plotted in indicated bins (k = 1000 bp). (B) De-novo motif analysis of FOXP1 ChIP-seq peaks in OCI-Ly1, OCI-Ly7 and OCI-Ly10 reveals the presence of several enriched motifs in the FOXP1-binding regions. Relative enrichment to control regions and percentage of peaks containing the motif are shown. (C) 1st column: the percentage of FOXP1-bound peaks in the corresponding cell line that contain the single most enriched forkhead binding domain, as identified by de-novo motif analysis in that cell line; 2nd column: the percentage of FOXP1-bound peaks in the corresponding cell line that contain any of the various FKHD motifs identified by de novo motif analysis in any of our cell lines; 3rd column: the percentage of the 2000 highest ranking FOXP1-bound peaks in the corresponding cell line that contain any of the various FKHD motifs identified in any of our cell lines. (D) Enrichment of apoptosis or cell death related GO terms among genes that are bound by FOXP1 within 20 kb of their TSS in the DLBCL cell lines. (E) ChIP-qPCR analysis in DLBCL cell lines using an antibody against FOXP1 and primers for pro-apoptotic genes (supplemental table III). (F) Tracks showing the locations of the FOXP1
ChIP-seq signal in the proximity of the TSS of HRK, EAF2. TP53INP1, and TP63, some of the pro-apoptotic genes that are repressed by FOXP1 in primary human B cells and DLBCL cell lines.

**Supplemental figure 3. Cell cycle analysis of transduced B cells**

Human primary B cells were transduced with FOXP1-IRES-YFP, BCL6-IRES-GFP or ctrl-IRES-YFP and cultured on CD40L-L cells IL-21 and IL-2. 12 days after transduction, cell cycle stage was determined by PI staining. The percentage of cells in each stage of the cell cycle was determined using the Watson Pragmatic model. Representative graphs of two independent experiments are shown.

**Supplemental figure 4. Analysis of BCL6 and BCL6/FOXP1 double transduced cells, cultured in the absence of CD40L-L cells.**

Memory B cells were sorted from human peripheral blood and co-transduced with FOXP1-IRES-YFP and BCL6-IRES-GFP (left graph) or transduced with control-IRES-YFP or BCL-IRES-GFP (right graph). Transduced B cells were cultured with IL-21, IL-2 and CD40L-L cells for the first three days, and subsequently with IL-21 and IL-2, either in the absence of CD40L-L cells. The percentages of each population was followed over time by FASC analysis and normalized to the percentage of each population at day 3 after transduction. Mean ± SD of two independent (left), or three independent (right) experiments are shown. (paired t test, *P<0.05, **P<0.01, ***P<0.001).

**Supplemental figure 5. The effect of CA-IKK2 and CD40L-L cells on the expression of NF-target genes, FOXP1 and pro-apoptotic genes.**
Memory B cells were sorted from human peripheral blood and transduced with either CA-IKK2-IRES-GFP or control-IRES-YFP and cultured without CD40L-L cells as of day 3 after transduction (left panel and C), or transduced with control-IRES-YFP and cultured with or without CD40L-L cells as of day 3 after transduction (right panel). Six days after transduction YFP or GFP positive cells were sorted. Gene expression levels of (A) NF-κB target genes (and IKBKB, the gene encoding for IKK2), (B) FOXP1, or (C) the 7 FOXP1-repressed pro-apoptotic genes were analyzed by quantitative RT-PCR. Expression levels were normalized to expression levels in control transduced cells. Mean ± SEM of three independent experiments are shown. (one sample t test, *P<0.05, **P<0.01).

Supplemental figure 6. siRNA-mediated knockdown of FOXP1 in DLBCL cellines does not affect growth, proliferation or survival.

DLBCL cell lines were nucleofected with control siRNA, siRNA against FOXP1 (siFOXP1-1, the siRNA also used in the rest of this study), or siRNA against FOXP1 also used by Craig et al8 (siFOXP1-2) against FOXP1. (A) Two or three days after nucleofection, RNA was isolated and gene expression levels were analyzed by quantitative RT-PCR. Expression levels were normalized to expression levels in control siRNA transduced cells. Mean ± SEM of at least two independent experiments are shown. (B) The number and percentage of live cells was determined on consecutive days by flow cytometry. TO-PRO staining was used to distinguish live and dead cells. Mean ± SEM of two independent experiments are shown Efficient knockdown was validated by quantitative RT-PCR analysis. (C) Cells were cultured in medium with either 10, 5, or 1% FCS immediately after nucleofection and the percentage of live cells were determined on consecutive days by flow cytometry. TO-PRO staining was used to distinguish live and dead cells. One experiment is shown. (D) Four days after nucleofection, caspase 3/7 activity was
determined by the caspase glo3/7 assay. Values were corrected for number of living cells as determined by FACS analysis. Mean ± SD of two independent experiments (one in the case of SUDHL6) are shown. (E) Cell lines were labeled with CFSE, 1 day before nucleofection. Three days after nucleofection, CFSE intensity was determined by flow cytometry. Representative graphs of two independent experiments are shown. (F) The cells were treated, directly after nucleofection with 5 μM of the NF-κB inhibitor BMS-345541. The number and percentage of live cells were determined on consecutive days by flow cytometry. TO-PRO staining was used to distinguish live and dead cells. Mean ± SEM of two independent experiments are shown.

Supplemental figure 7. FOXP1 expression levels in MBCs and DLBCL cell lines
(A) Quantitative-RT-PCR analysis for FOXP1 expression in DLBCL cell lines and primary human MBCs, transduced with CTRL-IRES-YFP or FOXP1-IRES-YFP IRES-YFP and cultured with CD40L-L cells, IL-21 and IL-2. Representative experiment is shown (B) Immunoblot analysis of FOXP1 expression in DLBCL cell lines. Representative blot is shown

Supplemental figure 8. FOXP1 overexpression in DLBCL cell lines, in the absence or presence of CA-IKK2.
DLBCL cellines were transduced with FOXP1-IRES-YFP, ctrl-IRES-YFP, or CA-IKK2-IRES-GFP (A) or -transduced with FOXP1-IRES-YFP and CA-IKK2-IRES-GFP (B,C) and the percentages of each population was followed over time by FASC analysis and normalized to the percentage of each population at day 3 after transduction. (A) Mean ± SD of two independent (OCI-Ly7), or one experiments are shown. (B) Mean ± SD of three (OCI-Ly7), or two independent experiments are shown. C) cells were labeled with eFluor 670 two days after
transduction. Four days after labeling, CFSE intensity was determined by flow cytometry. Representative graphs of two independent experiments are shown.

Supplemental table I
This table shows the genes that were reproducibly regulated by FOXP1 in 2 independent experiments, employing B cells from 2 different donors.

Supplemental table II
This table shows the genes within 20 kb of FOXP1 ChIP peaks, that are exclusively identified in ABC or GC DLBCL cell lines and GO term analysis of these genes. The 10 most significantly enriched GO categories are shown.

Supplemental table III
This file contains all the peaks identified with ChIP-seq using an anti-FOXP1 antibody. The peaks are ranked from high peak intensity to lower peak height.

Supplemental table IV
List of primers used for quantitative RT-PCR and ChIP-Q-PCR.

References


Supplemental figure 1

A

Group 1 n=207
Group 0 n=291

P=2.9E-4

B

high n=294
low n=294

P=4.4E-3

C

high n=235
low n=235

P=2.4E-3

high n=249
low n=249

P=4.1E-3
Group 1 n=96
Group 0 n=103

P=5.4E-3

Follow up in months

Group 1 n=86
Group 0 n=141

P=1.7E-3

Follow up in months

E  Group 0  |  Group 1
Supplemental figure 2

A

**OCI Ly1**

![Graph showing percentage of peaks vs. distance to closest TSS (kb) for OCI Ly1. The graph compares FOXP1 and random region.](image)

**OCI Ly3**

![Graph showing percentage of peaks vs. distance to closest TSS (kb) for OCI Ly3. The graph compares FOXP1 and random region.](image)

**OCI Ly7**

![Graph showing percentage of peaks vs. distance to closest TSS (kb) for OCI Ly7. The graph compares FOXP1 and random region.](image)

**OCI Ly10**

![Graph showing percentage of peaks vs. distance to closest TSS (kb) for OCI Ly10. The graph compares FOXP1 and random region.](image)
### OCI-Ly1

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## OCI-Ly7

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E

[Graphs showing % of input for OCI-Ly1, OCI-Ly3, OCI-Ly7, and OCI-Ly10 for different proteins like BIK, HRK, TP53, EAF2, HBB, FOXP1 IP, IgG IP, EAF2, TP63, HBB, BIK, HRK, RASSF6, HBB.]
Supplemental figure 3

LZRS  BCL6  FOXP1

G1  G2  G1  G2  G1  G2
70%  7%  68%  4%  74%  6%
S    H4  S    H4  S    H4
19%  24%  17%

PI
Supplemental figure 4

![Graph showing the relative percentage of transduced cells over days for different conditions.]

- ctrl
- BCL6
- FOXP1
- FOXP1+BCL6

Days: 0 5 10 15
Relative percentage transduced cells: 0.0 0.5 1.0 1.5 2.0

Significance levels:
- **: p < 0.05
- ***: p < 0.001
Supplemental figure 5

A

![Graph showing relative expression of various genes under different conditions.](image)

B

![Bar graph showing relative FOXP1 expression under different conditions.](image)

C

![Bar graphs showing relative expression of various genes under different conditions.](image)
Supplemental figure 6

A

Relative FOXP1 expression

B

Relative percentage viable cells

C

Relative percentage viable cells
**F**

**OCI-Ly3**

- Relative percentage viable cells

- Days: 0, 1, 2, 3

**U2932**

- Relative percentage viable cells

- Days: 0, 1, 2, 3

**OCI-Ly3**

- Relative number of cells

- Days: 0, 1, 2, 3

**U2932**

- Relative number of cells

- Days: 0, 1, 2, 3
Supplemental figure 7

A

Relative FOXP1 expression

B

75 kDa

50 kDa

FOXP1

B-actin

MBC-ctrl
MBC-FOXP1
OCI-Ly1
OCI-Ly7
SUDHL-6
OCI-Ly3
OCI-Ly10
U2932
Supplemental figure 8

Panel A shows the relative percentage of transduced cells for OCI-Ly1, OCI-Ly7, and SUDHL6 over 20 days, with three treatments: CTRL, FOXP1, and CA-IKK2. The y-axis represents the relative percentage of transduced cells, and the x-axis represents the number of days.

Panel B includes similar data for OCI-Ly1, OCI-Ly7, and SUDHL6, but with an increased scale on the y-axis, indicating the relative percentage of transduced cells for each treatment over 20 days.

Panel C displays flow cytometry histograms for OCI-Ly1, OCI-Ly7, and OCI-Ly10, stained with eFluor 670, showing the distribution of cells across different treatments: FOXp1, CA-IKK2, and FOXP1+CA-IKK2.

The histograms indicate the number of cells on the x-axis and the fluorescence intensity on the y-axis.