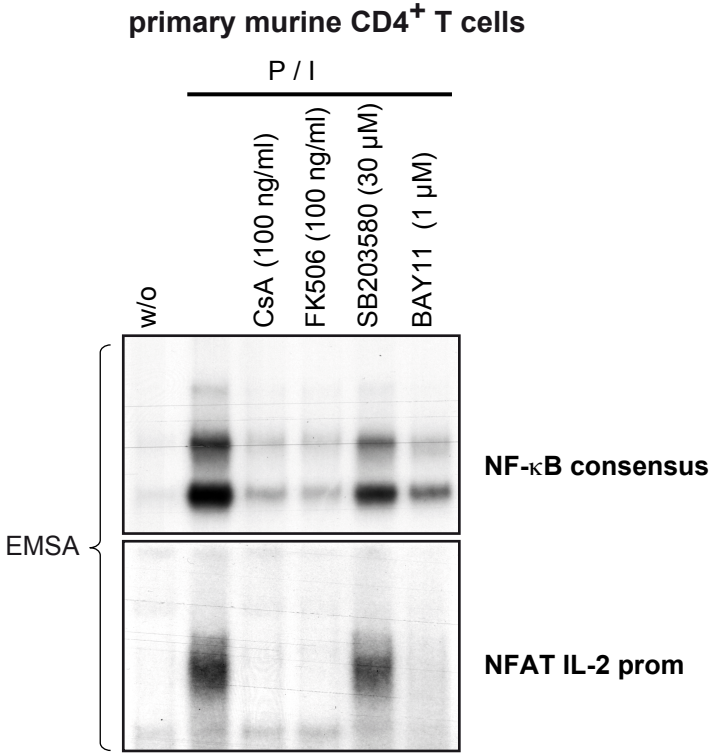
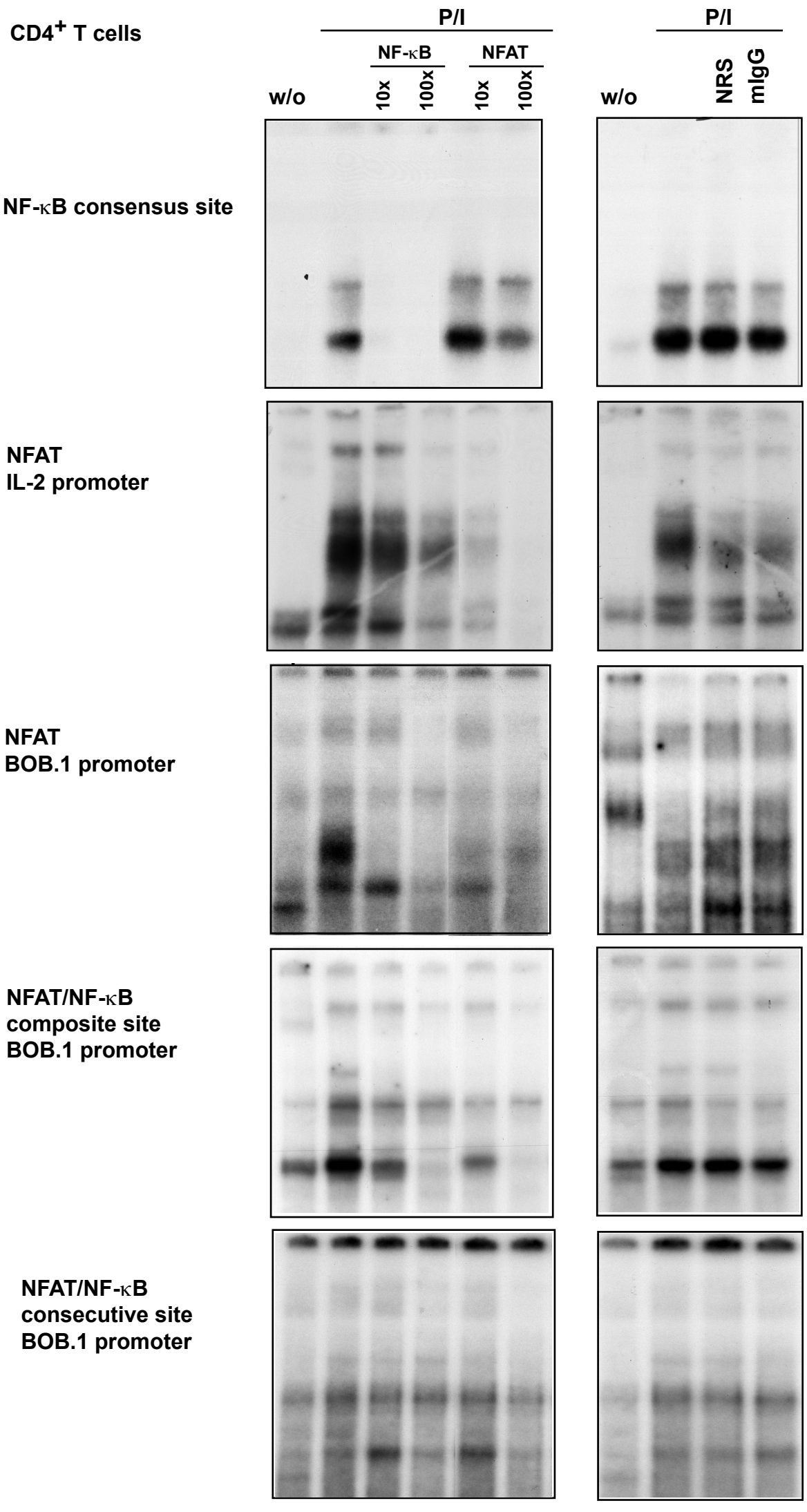
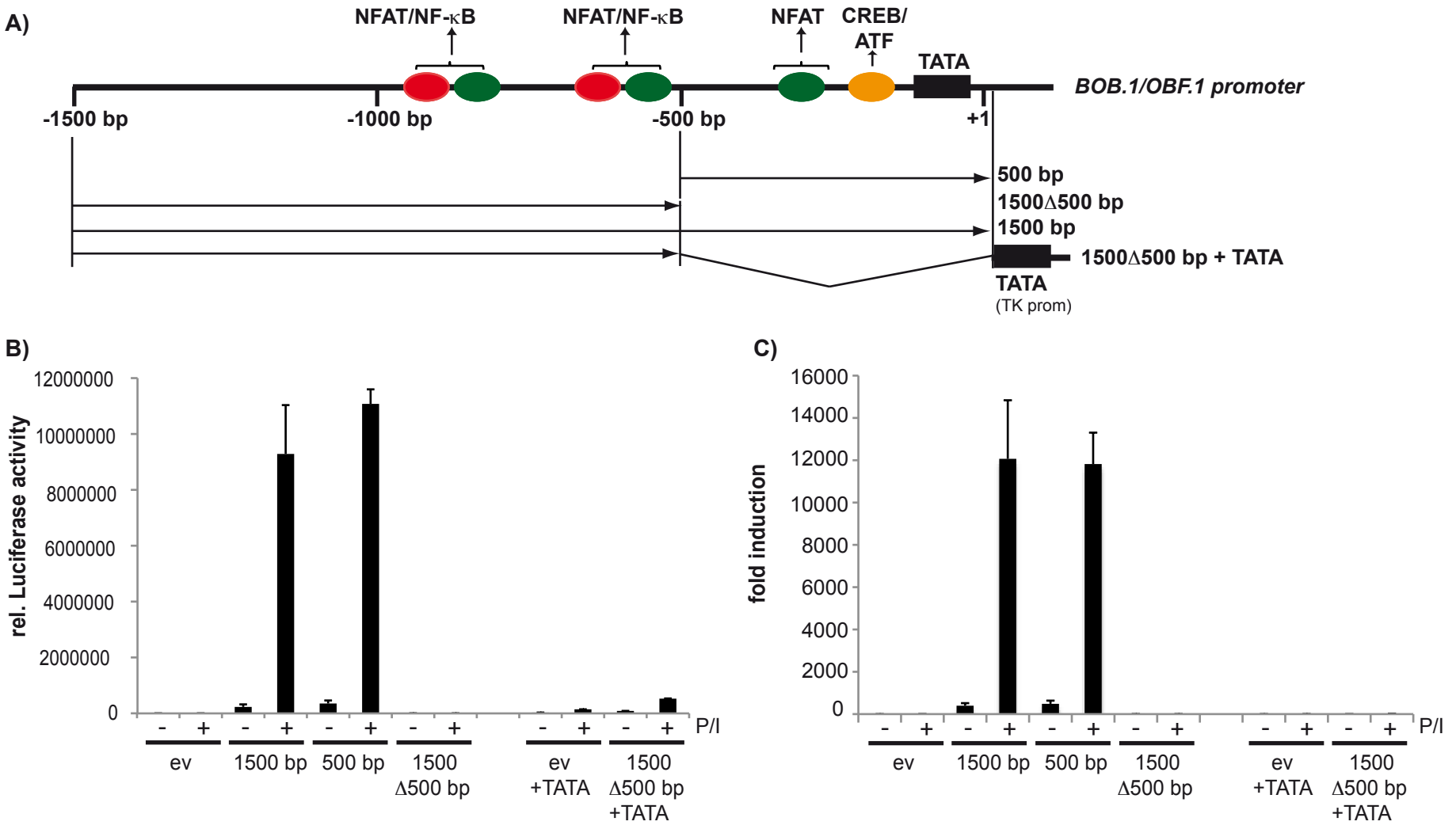
Supplementary Figure 1



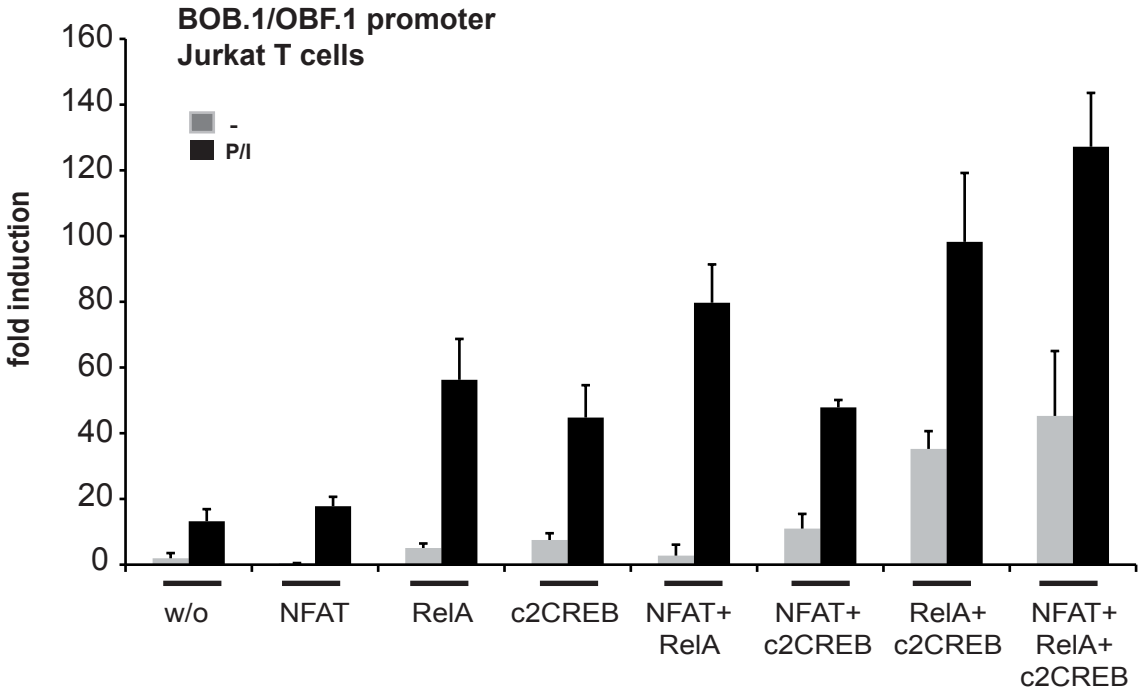


Supplementary Figure 2

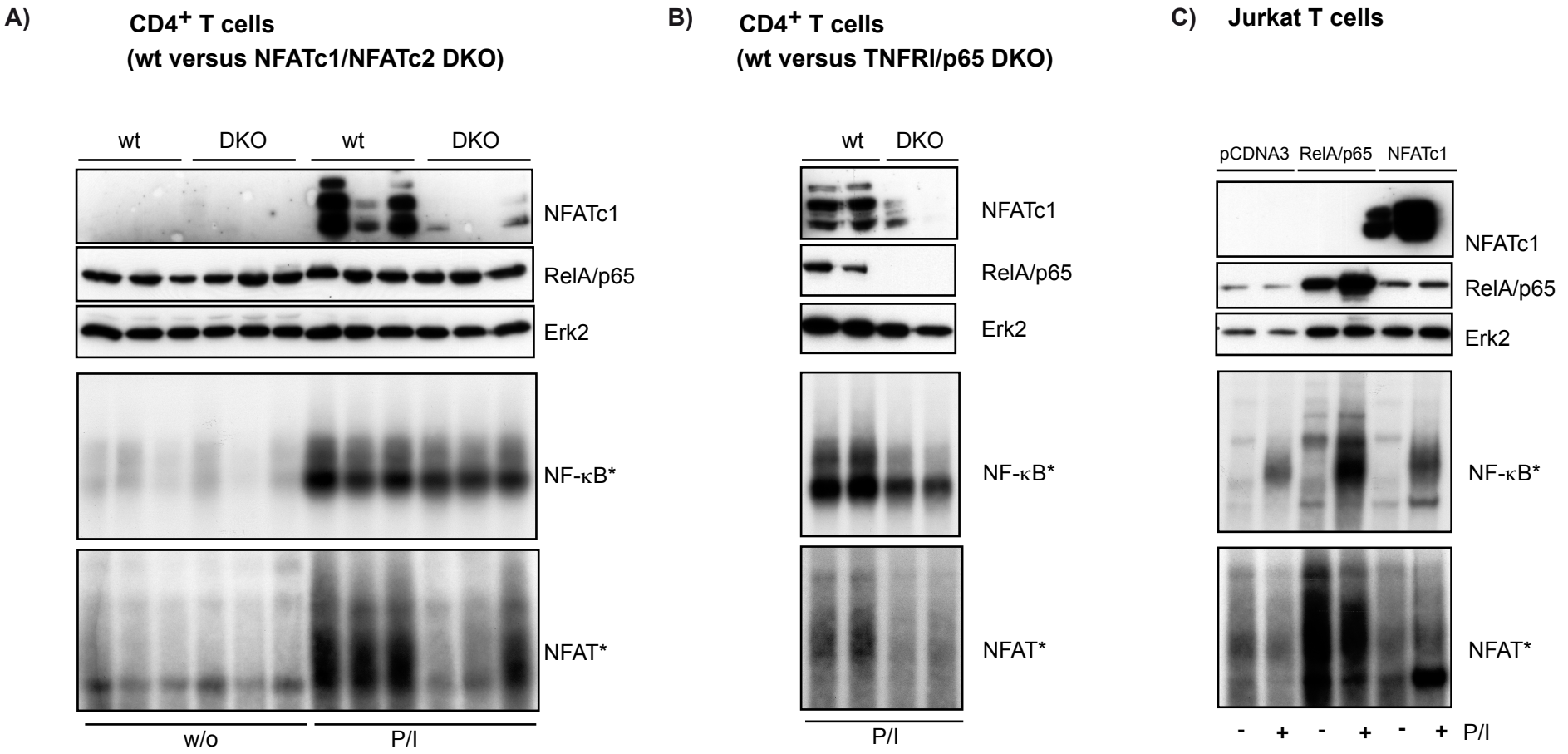
Supplementary Figure 3



Supplementary Figure 4



Supplementary Figure 5



probe	forward	reverse
NF-κB	gcggGCCTG**GG**AAAGTCCCCTCAACT	gcggAGTTGAGGGGACTTTCCCAGGC
NF-κB-Mut	gcggGCCTG**tt**AAAGTCCCCTCAACT	gcggAGTTGAGGGGACTTT**aa**CAGGC
NFAT-IL2 prom	ggcgACCCCAAAGA**GG**AAAATTTGTTTCATACAG	ggcgCTGTATGAAACAAATTTT**CC**TCTTTGGGGT
NFAT-IL2 prom-Mut	ggcgACCCCAAAGA**tt**AAAATTTGTTTCATACAG	ggcgCTGTATGAAACAAATTTT**aa**TCTTTGGGGT
NFAT-Bob prom	ggcgGGCGGTGTTGCT**G**AA**G**AAAAAAAAA**G**TACA	ggcgTGTACTTTTTTTTTCTTCAGCAACACCGCC
NFAT-Bob prom-Mut1	ggcgGGCGGTGTTGCTGAA**t**AAAAAAAAA**G**TACA	ggcgTGTA**C**TTTTTTTTT**a**TTCAGCAACACCGCC
NFAT-Bob prom-Mut2	ggcgGGCGGTGTTGCTGAA**G**AAAAAAAAA**t**TACA	ggcgTGTA**a**TTTTTTTTT**C**TTCAGCAACACCGCC
composite NFAT/NF-κB-Bob.1 prom	ggcgGAGGGCA**GG**TGTTT**CC**TCCCATGGTG	ggcgCACCATGGGA**GG**AAACA**CC**TGCCCTC
composite NFAT/NF-κB-Bob.1 prom-Mut1	ggcgGAGGGCA**tt**TGTTT**CC**TCCCATGGTG	ggcgCACCATGGGAGGAAACA**aa**TGCCCTC
composite NFAT/NF-κB-Bob.1 prom-Mut2	ggcgGAGGGCA**GG**TGTTT**aa**TCCCATGGTG	ggcgCACCATGGGA**tt**AAACACCTGCCCTC
consecutive NFAT/NF-κB - Bob.1 prom	ggcgAGTGACCCCTG**GG**AATCGCATTT**CC**CGTCGAGC	ggcgGCTCGACG**GG**AAATGCGATT**CC**CAGGGGTCACT
consecutive NFAT/NF-κB - Bob.1 prom-Mut1	ggcgAGTGACCCCTG**tt**AATCGCATTT**CC**CGTCGAGC	ggcgGCTCGACGGGAAATGCGATT**aa**CAGGGGTCACT
consecutive NFAT/NF-κB - Bob.1 prom-Mut2	ggcgAGTGACCCCTG**GG**AATCGCATTT**aa**CGTCGAGC	ggcgGCTCGACG**tt**AAATGCGATTCCCAGGGGTCACT

Supplementary Table 1

gene	forward	reverse
murine *Bob.1/Obf.1* (*Pou2af1*)	catgctctggcaaaaatcc	actcgaacaccctggtatgg
murine *Oct2* (*Pou2f2*)	ggcccaactcatgctgac	agctggaggagttgctgtatg
murine *Rpl13*	cctgctgctctcaaggttgt	ggtacttccacccgacctc
murine HPRT	cctaagatgagcgcaagttgaa	ccacaggactagaacacctgctaa
human *BOB.1/OBF.1* (*POU2AF1*)	cacagctccggagcaagcc	ccttccacagagagagtgtgg
human *OCT2* (*POU2F2*)	atggagaaggaagtgatccg	ttgatgcgtttctccttctg
human *RPL13*	cggaccgtgcgaggtat	caccatccgctttttcttgtc
Human HPRT	gaccagtcaacaggggacat	cttgcgaccttgaccatctt

Supplementary Table 2

ChIP primer	forward	reverse
NFAT/κB BOB.1 promoter	gcaacaccgagaacgaggag	catacagttccacactatcag
NFAT BOB.1 promoter	ggcctcctgtgctgtatggtgg	ggcccgaattcccgctgagag
Bob.1 promoter control region	cactggagaatgaaggatccagaccc	ctcgtaggctccattcaagtgaccc

Supplementary Table 3

in vitro mutagenesis primer	**forward**	**reverse**
ivM NFAT site	gcggtg ttgctgaata aaaaaaatta cagctctgcc tgagg	cctca ggcagagctg taatttttt tattcagcaa caccgc
ivM consecutive NFAT/NFkB site	gaatagt gacccctgtt aatcgcattt aacgtcgagc tgggacc	ggtccca gctcgacgtt aaatgcgatt aacaggggtc actattc
ivM composite NFAT/NFkB site	ggct tgaagagagg gcatttgttt aatcccatgg tgagaatagt gacc	ggtc actattctca ccatgggatt aaacaaatgc cctctcttca agcc
ivM CREB site	c tgaggtagga ggatgtgatt gctcggcccc tctcagc	gctgaga ggggccgagc aatcacatcc tcctacctca g

Supplementary Table 4

LEGENDS TO SUPPLEMENTARY FIGURES AND TABLES

Supplementary Figure 1**: CsA, FK506 and Bay-11 affect the binding of NFAT and NF-κB to DNA.** A) Primary murine $CD4^+$ T cells were treated as indicated. After 18 h of stimulation cells were harvested, protein extracts were prepared and analyzed by EMSA for their ability to bind to DNA bearing a consensus NF-κB or NFAT motif.

Supplementary Figure 2. **Consensus NFAT and NF-κB sites compete against complex formation observed on newly identified NFAT/NF-κB site of the BOB.1OF.1 promoter.** In order to investigate the potential binding of NFAT and NF-κB factors to the newly identified NFAT/NF-κB site of the BOB.1/OBF.1 promoter observed in experiments depicted in Figure 2 competition experiments were performed using 10 or 100 fold molar excess of non-labeled consensus NFAT or NF-κB sites together with labeled sites representing sequences of the BOB.1/OBF.1 promoter and whole protein extracts of unstimulated or with P/I treated $CD4^+$ T cells. In addition, to prove the specificity of antibody binding observed in supershift experiments (Figure 2) control experiments were performed using murine IgG antibodies (mIgG) or normal rabbit serum (NRS).

Supplementary Figure 3. **The *BOB.1/OBF.1* promoter spanning 500 bp is necessary and sufficient for full inducible activity in T cells.** A) Schematic representation of the BOB.1/OBF.1 promoter. The positions of the analyzed *cis*-elements as well as of the promoter fragments used in reporter assays are indicated. B and C) Jurkat T cells were transiently transfected with the empty vector (ev) or with reporter constructs bearing either the 1500 bp or the 500 bp *BOB.1/OBF.1* promoter construct or a deletion mutant of the longer version were the first 500 bp are missing (1500Δ500 bp). Additionally, Jurkat cells were transfected with an empty vector containing a TATA box (ev + TATA) or with a vector, were the 1500Δ500 bp BOB.1/OBF.1 promoter construct was cloned in front of a TATA-box (1500Δ500 bp +TATA). The relative luciferase activity (B) or the fold induction (C) of different promoter fragments was determined without or after stimulation of cells with P/I, were in the second case the fold induction was determined relative to the luciferase activity of the empty vector without stimulation that was set to one. Shown are the mean values ± s.d. of four independently performed experiments.

Supplementary Figure 4. **A constitutive active version of CREB transactivates together with NFAT and NF-κB the BOB.1/OBF.1 promoter.** Jurkat T cells were transiently transfected with the 1500 bp BOB.1/OBF.1 promoter construct either alone or together with expression vectors for NFATc1, RelA/p65 or the constitutive active version of CREB (c2CREB) or with combination of those as indicated. Transfected cells were either left untreated or stimulated with P/I. Next day the cells were harvested to determine the relative luciferase activity. The relative luciferase activity resulting from the transfection of the 1500 bp promoter construct alone without induction was set to one. The fold induction relative to this value was calculated and is depicted. Shown are the mean values ± s.d. of three independently performed experiments.

Supplementary Figure 5. **Modulation of NF-κB activity influences the expression and consequently the binding capacity of NFAT.** Murine wildtype $CD4^+$ T cells, $CD4^+$ T cells deficient for NFATc1 and NFATc2 (DKO in (A)) or deficient in TNFRI and p65 (DKO in (B)) were analyzed for protein expression of NFATc1 and RelA/p65 in Western Blots. The analyses of ERK2 protein expression served as loading control. The NFAT and NF-κB binding activity was assayed in EMSA experiments using labeled NFAT and NF-κB consensus sites as probes. C) Jurkat cells were transiently transfected with RelA/p65 or NFATc1 expression vectors and subsequently analyzed as described in A) and B).

Supplementary Table 1. Sequences of oligomers bearing different potential as well established transcription factor binding site used after annealing as labeled dsDNA probes in EMSA.

Supplementary Table 2. Sequences of primers used for the analyses of mRNA expression of human as well as murine *BOB.1/OBF.1* and *OCT2* as well as the house-keeping gene *RPL13* by quantitative RT-PCR.

Supplementary Table 3. Sequences of primers used for the analyses of enriched genomic DNA fragments after chromatin immunoprecipitation.

Supplementary Table 4. Sequences of primers used for in vitro mutagenesis of the 1500 bp BOB.1/OBF.1 promoter.