Gene Name	Forward Primer	Reverse Primer
ACTB	CTGGAACGGTGAAGGTGACA	AAGGGACTTCCTGTAACAATGCA
ACVRL1	ACATGAAGAAGGTGGTGTGTGTGG	CGGGCAGAGGGGTTTGGGTA
ADAMDEC1	GGGGCCAGACTACACTGAAACATT	ACCCGTCACAAGTACTGATGCTG
AHI1	GTCCAAAACTACCCCATCAAGGCT	GCAGCACAGGAACGTATCACCT
ANGPT2	TGGCAGCGTTGATTTTCAGAGG	GCGAAACAAACTCATTTCCCAGCC
ANPEP	TGAAGAAGCAGGTCACACCCCT	AACTCCGTTGGAGCAGGCGG
APOA1	GCCGTGCTCTTCCTGACGG	TGGGACACATAGTCTCTGCCGC
ATXN7	CACCGCCCACTCTGGAAAAGAA	GGGTGCAGGGCTTCTTGGTG
B2M	TGCTGTCTCCATGTTTGATGTATCT	TCTCTGCTCCCCACCTCTAAGT
BAG4	AGGTTCCAGGATATCCGCCTT	TCGGTCCTGATTGTGGAACACT
BCL2	ACAACATCGCCCTGTGGATGA	CCGTACAGTTCCACAAAGGCAT
BCL2L14	GCTCAGGGTCAAAGGACGTTGG	TCAGCTACTCGGTTGGCAATGG
BCL7A	GAACCATGTCGGGCAGGTCG	CCCATTTGTAGATTCGTAGGGATGTGT
BIN1	TGCTGTCGTGGTGGAGACCTTC	GCCGTGTAGTCGTGCTGGG
BIRC3	TGCTATCCACATCAGACAGCCC	TCTGAATGGTCTTCTCCAGGTTCA
BIRC5	TTCTCAAGGACCACCGCATCT	AGTGGATGAAGCCAGCCTCG
BLK	TCGGGGTCTTCACCATCAAAGC	GCGCTCCAGGTTGCGGATGA
BTRC	CCAAATGTGTCATTACCAACATGGGC	GCAGCACATAGTGATTTGGCATCC
BUB3	CGGAACATGGGTTACGTGCAGC	CCAAATACTCAACTGCCACTCGGC
CAGE1	TCCAAAATGCACAGTCTTCTGGCT	GGAGGCTCTTCAGTTTTTGCAGC
CASP1	CCTGTTCCTGTGATGTGGAGGAAA	GCTCTACCATCTGGCTGCTCAA
CASP3	AGCGAATCAATGGACTCTGGAATATCC	GTTTGCTGCATCGACATCTGTACCA
CCL5	TCATTGCTACTGCCCTCTGCG	ACTGCTGGGTTGGAGCACTTG
CCL18	CCCTCCTTGTCCTCGTCTGCA	GCACTGGGGGCTGGTTTCAG
CCL26	TTCCAATACAGCCACAAGCCCC	GGATGGGTACAGACTTTCTTGCCTC
CCND2	TCAAGTGCGTGCAGAAGGACAT	CTTCGCACTTCTGTTCCTCACA
CCND3	TGGCTGCTGTGATTGCACATGA	GATGGCGGGTACATGGCAAAGG
CCR3	ACGCTGCTCTGCTTCCTGG	TCCTCAGTTCCCCACCATCGC
CCR4	AGCATCGTGCTTCCTGAGCAA	GGTGTCTGCTATATCCGTGGGGT
CCR7	AGACAGGGGTAGTGCGAGGC	CTGGAAAATGACAAGGAGAGCCACC
CD164	AAACCTGTGAAGGTCGAAACAGCT	AGTCTGTCGTGTTCCCCACTTGA
CD1D	CACGTTCAGCGACCAGCAGTG	CTCACAGCCAGCGGACACCT
CD22_A	CATCCTCATCCTGGCAATCT	CTCTGCATCTCCAGTTCGTG
CD22_B	TTTTTGAGCACCCTGAAACC	CGGATACCCATAGCAGGAGA
CD274	ACCAGCACACTGAGAATCAACACA	GTCCTTTCATTTGGAGGATGTGCCA
CD52	CCTCCTACTCACCATCAGCCTCC	TGCTACCAAAGCTGCCTCCTGG
CD7	GGACAACCTGACTATCACCATGCA	TCCGAGCATCTGTGCCATCCTT
CD70	TGGGACGTAGCTGAGCTGCAG	GTCGTGGAGGAGCAGATGGC
CDKN1C	AGATCAGCGCCTGAGAAGTCGT	CTCGGGGCTCTTTGGGCTCT
CDKN2A	GGCACCAGAGGCAGTAACCAT	AGCCTCTCTGGTTCTTTCAATCGG
CDKN2B	GGGGCTGGAACCTAGATCGCC	CGGTCGGGTGAGAGTGGCA
CDO1	ACAGTCCACCTTTTGATACATGCCA	GCCCCTTAGTTGTTCTCCAGCG
CHD1	AGACCGACATCAGGGAGATTCTTACA	CCTGTGATCATCCAGTTTTCTGTGTTTC
CHD7	GGCACAGCTCCACCCATCAC	CTGAGTCATATCCGGCACTGGTTT
CLU	CGCCACAACTCCACGGGCTG	GTCAACCTCTCAGCGACCTGGA
CNOT3	CGTCCGTCTCCAAGAGAGTATGAAGA	CAAACTGCTCCACGCCCTCG
CR592140	ACCAGTGAAATTGACCTGCCCG	GCCCCAACCGAAATGTTTAACGC
CST6	CTGACGATGGAGATGGGGAGCA	GCCAGGGAACCACAAGGACC
CTAG1A	CGTGCCAGGGGTGCTTCTGA	TGCGTGATCCACATCAACAGGG

CTAG2	CAGGGGCAGCAAGGGCC	GCGACGAGAAAGGCATCGTGAT
CTAGE1	TCCTTACCGTCCCCCAAGACCT	GCTGTCGTTCTGGATGTTCAGCA
CTLA4	AGCTGAACCTGGCTACCAGGAC	ACACAAAGCTGGCGATGCCT
CTNNB1	TGGATACCTCCCAAGTCCTGTATGAG	TGCCCTCATCTAATGTCTCAGGGA
CXCL9	TGCTGGTTCTGATTGGAGTGCAA	AGGAAGGGCTTGGGGCAAATTG
CXCL11	CAGCAGCACCAGCAGCAACA	TGCAAAGACAGCGTCCTCTTTTGA
DDX53	TGGAAGAGGGCGGAGGCTAATC	AAAGCAGAGTGGTGGTTCACGG
DMAP1	GCGCGGATGTACGGGACATTC	CCTCGGGCCTCTTGAAAGTCAG
DMC1	TGCAATGTCAAAGGACTCTCAGAAGC	CCCGGTGGTGATATGGAAAACCA
DNM3	CTCCAGCCAACACTGATCTTGCA	CTGGCATCCGTTCCTTCATCCA
DPP4	TGGTATCAGATGATCTTGCCTCCTCA	CCACTTCCTCTGCCATCAAAGCT
E2F1	ATGGTGATCAAAGCCCCTCCT	TCGATCGGGCCTTGTTTGCT
E2F4	AGATACCCTCTTGGCCATCCG	GTGAATCTGGTACTTCTTCTGCCC
EED	TGCGGCCAAGAAGCAGAAGC	TGCATTTGGCGTGTTTGTAGGTG
EP400	TGCCCCCACCAAACCACAGA	TGCTTTCCTCAGCTCCGCAATG
EPHA4	GGCAGATGGTGAATGGCTGGT	GAGTAGCTGTGGGGTGGGCA
ESRRB	CGGGGACATTGCCTCTGGCTA	TGATCTCGCACTCGTTGGTGGC
EZH2	AAAATTATGATGGGAAAGTACACGGGGA	CTTCTCTTTCTTCAGGATCGTCTCCATC
FAS	AACCATGCTGGGCATCTGGA	GTTGATGTCAGTCACTTGGGCA
FASTK	AGGAAACGCAACTCAGCAGCAA	TGCCACCCCTGCTTCCCGA
FCRL3	CCCAGCACAGTCATGGAGTGAG	GTGTCATCCTCGTGATAAAACCAGTACA
FLT4	CCTGACACGCTCTTGGTCAACA	CCGGTCATCCCACACCACCT
FOSL1	CGGAGGAAGGAACTGACCGACT	TTCCAGCACCAGCTCTAGGCG
FOXP3	AGCTCCTACCCACTGCTGGC	TGCCCTGCCCTTCTCATCCAG
FYB	CCTCCCTTGTTTACCTTGGGTCC	GTGGAGGTGGTGGCAGGGAA
GAGE6	ACAGCCTCCTGAAATGATTGGGC	TGCTCCCTCATCCTCTCCCTCC
GATA3	GGCAACCTCGACCCCACTGT	CGTCCCTGCTCTCCTGGCTG
GATA6	TGAACCCGTGTGCAATGCTT	TTTCATAGCAAGTGGTCTGGGC
GNLY	TGGTCTTCTCTCGTCTGAGCCC	CCCAGCTCCTGTGTTTTGGTCA
GTSF1	GCAGACCAGCACCCCATTTGTC	GGCAGAGATTTGGGAACTCGCA
HCK	GGGGTGCATGAAGTCCAAGTTCC	CTGGTGTGTTGCTGTTGTGGCT
HDAC1	CGATGGCCTGTTTGAGTTCTGTCA	TCGGACTTCTTTGCATGGTGCA
HDAC2	TGGCGTACAGTCAAGGAGGCG	AGCAAGTTATGGGTCATGCGGATT
HIF1A	ACCCTAACTAGCCGAGGAAGAACTATGA	AGGTGGTTTCTTATACCCACACTGAGG
HMBS	GCTTCACCATCGGAGCCATCT	TGGCAGGGTTTCTAGGGTCTT
IFI35	TGGGGCTGAGAGAGACCACAG	CTGAAGGGCGTGGAGGGC
IFNG	GCATCGTTTTGGGTTCTCTTGGC	CCGCTACATCTGAATGACCTGCA
IGFL2	TCTGTCTCCTCCTCTTGTGTCCAA	CGGGTCTCGCTCAGGGACA
IK	CCCATGGACGTTGACAAAGGA	CAGCAGACCCAGCAAACTTTTCA
IL1F7	GGACAAAGTCATCCATCCCTTCAGC	CCGACTCCAGCATGTTCCAGG
IL1RN	GCCGACCCTCTGGGAGAAAAT	TGGTTGTTCCTCAGATAGAAGGTCT
IL2	CCAAACTCACCAGGATGCTCACA	ACGTTGATATTGCTGATTAAGTCCCTGG
IL2RA	GAAAGACCTCCGCTTCACTGCC	GGATCTCTGGCGGGTCATCGT
IL4	GCAGTTCCACAGGCACAAGCA	GGTTGGCTTCCTTCACAGGACA
IL5	GCTGATAGCCAATGAGACTCTGAGG	TCCACAGTACCCCCTTGCACA
IL7R	AGTGAATGGATCGCAGCACTCAC	AAATTCAGGCACTTTACCTCCACGA
IL9	TGACCAGTTGTCTCTGTTTGGGC	TGGGTATCTTGTTTGCATGGTGGT
IL10	AGGAGGTGATGCCCCAAGCTG	GCCTTGCTCTTGTTTTCACAGGG
IL12A	TGGCAGTTATTGATGAGCTGATGCA	AGCATGAAGAAGTATGCAGAGCTTGA
IL13	GCATGGTATGGAGCATCAACCTGA	CCTCTGGGTCTTCTCGATGGCA

IL15	AGGCATTGTGGATGGATGGCTG	AACACAAGTAGCACTGGATGGAAATACT
IL17A	GTCAACCTGAACATCCATAACCGGA	GCACTTTGCCTCCCAGATCACA
IL17F	TCACGTAACATCGAGAGCCGC	TGGAGATGTCTTCCTTTCCTTGAGCA
IL17RA	TGCCCACACCCAACAAGGAGA	ACTCAAACCTGACGCACAAACGT
IL17RB	CGAGCTTCAGTGGTGATTCCAGT	GCCTGTTTGTGGGCAGAGCA
IL17RC	ACTGGACCGCAGATCATTACCTTG	CTGAAGGGGCAGATGTTCGTCC
IL18	TCATTGACCAAGGAAATCGGCCTC	TCACACTTCACAGAGATAGTTACAGCCA
IL21	TCTGCCAGCTCCAGAAGATGTAGA	TCTCCCTGCATTTGTGGAAGGTG
IL21R	AAGGAAGGCTGGAACCCTCACC	GGGGCATGAAGAACCGCTCAG
IL22	CCCTTGAAGAAGTGCTGTTCCCT	TCAGCTTTTGCACATTCCTCTGGA
IL23A	TGCTCCCTGATAGCCCTGTGG	TTTGAAGCGGAGAAGGAGACGC
IL23R	AGTGCCCAAGACCATAATTTATTGGGAT	TCCAAGTAGAATTCTGACTGTTGCACA
IL26	TCCTGTGCTTCATCAGCTAGAGAGA	GGCTTTGGTTTACTGACTGCTTTCC
IL32	CCTTGGCTCCTTGAACTTTTGGC	CATTCGGGCCTTCAGCTTCTTCA
IRF1	CGCTGTGCCATGAACTCCCTG	AGCATCTCGGCTGGACTTCGAC
IRF3	CCAGCCAGACACCTCTCCGG	GCAGGGCTCAGGGGCTACAG
IRF4	TATGCTTGTGCCCCACCTGAGT	ACGTGGTCAGCTCCTTCACGA
IRF7	CTGGGCTTCGGGCAGGAC	AGGGAAGACACACCCTCACGC
ITK	GGCTCAACAAGGACAAGGTGGC	TCCAGGCACACCCCATACAGC
JARID2	AGGCTAGTGGAAGAGAAGGACTGC	CCTGTGTTATTGGGGAGGACGG
JUNB	GAACGCCTGATTGTCCCCAACA	CGAAGCCCTCCTGCTCCTCG
KAT5	TCCTGAGCGTGAAGGACATCAGT	GCCTCTTTCTTGGGGAACTGGATC
KIR3DL2	TGCATGTTCTGATTGGGACCTCAG	TTCACTGTTCTGTCCCCCGCA
KIT	GCACCGAAGGAGGCACTTACAC	GCTGCCACACATTGGAGCATG
KLF4_	ATCTCAAGGCACACCTGCGAA	ATCTGAGCGGGCGAATTTCCAT
KLHDC5	GGATGCGTGGAATTTTGTGGCG	CATGTCGGAAGAGGGGCAGTAC
LCE2B	TGCTCCTGCGTGTGACCAGG	GGGGCAGGCATTTAGGGGGAC
LCK	GGAGATCTGGGCTTTGAGAAGGGG	GCCACAAAATTGAAGGGGATGAAGC
LEF1	AGCGAATGTCGTTGCTGAGTGT	AGCTGTCTTTCTTTCCGTGCTAATTCA
LIF	TGCCATACGCCACCCATGTCA	CAGGTTGTTGGGGAACGGCT
LOR	CTCTCCTCACTCACCCTTCCTGG	CCACCGCCGCCAGAGGTCTT
LTA	ACAGCACCCTCAAACCTGCT	CGGTCCGTGTTTGCTCTCCA
LTBP4	CGGCATCTGTACCAACACCGAC	CTGCGACCCGCACAGGG
MAGEA9B	AACCAGGAGGACAGGAGCCC	CATCAGGCCCAAGTCCTCTCCT
MAL	CTTGCCCGACTTGCTCTTCATCT	CCACGAAGCAGAACACAGACACG
MAX	ACGGGCTCATCATAATGCACTGG	TGTGGCTTTGTCTAGGATTTGGGC
MCL1	AAGGACAAAACGGGACTGGCT	CACATTCCTGATGCCACCTTCT
MDM2	CCGTGAAGGAAACTGGGGAGTC	CGAAGCTGGAATCTGTGAGGTGG
MIR155	CTGTTAATGCTAATCGTGATAGGGGT	AATGCTAATATGTAGGAGTCAGTTGGAG
MIR203	TGGGTCCAGTGGTTCTTAACAGTTC	TCGCTGTCGCCGCGC
MIR205	AAAGATCCTCAGACAATCCATGTGCTT	TGTCAGCTCCATGCCTCCTGAA
MMP2	CACTGCGGTTTTCTCGAATCCA	TTACCGTCAAAGGGGTATCCATCG
MMP9	TTCGACGATGACGAGTTGTGGT	CGAAGATGAAGGGGAAGTGGCA
MMP12	GCCGTAATGTTCCCCACCTACAA	TCAGGATTTGGCAAGCGTTGGT
MOS	TGCTGTGCTTCCAGACACCCT	CCTGCTTGGTAGTCATTTGCCAGA
MPZL2	TGGGTTTCCCTCATGTATGGCAAG	CATTAACAGCCTCCAGCACCCG
MRC2	CAGGACTACGGCAAAGACGAGC	GCCTCCCTCCACGACAGCGT
MTF2	ACTGAGGGAACTGCACATTCATCC	GGCCAAGATCTTCCTGTACGCG
MXI1	CCCGGCACACAACACTTGGTTT	CGCCACTTTAAAAATCTCTGTTCTCGTT
MYB	GTCATGTTCCATACCCTGTAGCGTT	CTGCTATCCCCTCATTCAAGCACA

MYC	GAGACACCGCCCACCACCAG	TCCAGCAGAAGGTGATCCAGACT
NAIP	TTCCTGGGTCCAGAGAGAATTACC	TCCAGCCGTAGTTCTTCGTAAGC
NANOG	TGGATCCAGCTTGTCCCCAAA	AGGCCCACAAATCACAGGCATA
NAV3	ACCTCTGGTTTCCCCTTCTGCC	AGTCTTGGGCTGGGATGCTGTT
NEDD4L	AGGATCTCGGACCAGCCCTCA	TGACACTGCATGACCTCAACCTTG
NFKB1	TGCAACTATGTGGGACCAGCAA	AGTGTTTTCCCACCAGGCTGT
NFKB2	GAGATGGAGGAGCTGGGGTTGG	CAGAAGGAGGCGGGTGAGGG
NKG7	GCCTCCACACCCCCAGATCC	TCTGCTCACAAGGTTTCATAGCCAG
NKIRAS2	TGCTCAAGAAGGAGATTGACAAATCCA	ACACCTCCCACAGCTTCACCTT
NOTCH1	AGCTGGACCCCATGGACG	GGTGGCACTCTGGAAGCACT
NR0B1	AGGGGACCGTGCTCTTTAACCC	AGTTCGATGAATCTGTCATGGGGC
NUB1	GTTGCAAGGCAATTGAGCGTGG	GCCAGTGATGTGCAATCGGGTC
PALM2-AKAP2	TGCCGAAGAGGAGGAAGCCAG	TCTGGGACTCTTCACTTTCTAGCGT
PDCD1	TCGTCTGGGCGGTGCTACAA	GGTGAAGGTGGCGTTGTCCC
PHC1	CCAAACACCAGCACTACACAGCA	GCACAGATTGGGTCAAGGTGGT
PIP5K1B	ACACTGTTTCTGTTCATAGACCAAGCT	AGGGCGGCGATTGAATTGCAC
PLK1	AGTACCTGCACCGAAACCGAGT	GGGTCTTCTTCCTCTCCCCGTC
PLS3	ACTCTCTTGGTGTCAATCCTCACGT	TCCCAGTTTCGGGTATGGAGGT
POU2AF1	GGGGCTCAGATAAGTCCTCTCTGG	GTGGTTTGCCCACAGCTAATTTTCA
POU5F1	TGCAAAGCAGAAACCCTCGT	TCGGGCACTGCAGGAACAAAT
PRDM2	ACACTACTGAGCCTGTGGCGG	TGCCTTTTAAAATTGGTTTAGTGGCCC
PSMD3	GGCCCTAGACCTTGTAGCCGC	CATGCCGAAGCGTAGCTGTCC
PSORS1C2	CAGCTTTGGGGGCCAGTACAT	CCTCTGCGGGTGGGTGAGAG
PTEN	ATTCCCAGTCAGAGGCGCTAT	TCATCTTGTGAAACAACAGTGCCA
PTGS2	ACAGGCTTCCATTGACCAGAGC	ACCATAGAGTGCTTCCAACTCTGC
PTPN6	GGGCATGGTGCAGACGGAGG	TGGCTGGGGGATAGGTGATGTT
PTPN7	CCAGGACATGAAAGAGTGCCCAG	AGCTGATTCTGGTGTCTGATGGTCT
PTPRG	TGGAAGCCATTCCTGTCAAACAGT	TGCAGTGATGTTCATATCAGCAGTACA
RAC2	GCCAATGTGATGGTGGACAGCA	TGACGAGGGAGAAGCAGATGAGG
RASA1	ACGATAGCAGAAGAACGCCTCA	AAAGTACAAAGGACCCTGGCCTC
RB1	CAGATGGTATGTAACAGCGACCGT	TCAGTGGTTTAGGAGGGTTGCTTC
REC8	TGATGGAGACCCTAGAAGATGCTCC	ACTCTCTCTGGGATTGCAGCCT
RHOF	GCGTGACCGTTGGCAGCA	CGTTGTCGTAGCTGGTGGGATT
RNF2	GCCTCATCCCACACTTATGGAAAAAGA	AGTTCTTCTAAAGCTAACCTCACAGCC
SALL4	ACCCCAGCATCTGGCTAAAACAC	GTGGCTTCATCCTCACTCGCCA
SCPEP1	TGCAGAGCAAGTACTGAATGCCG	TCCATTGTAGACGTGGGAGTGCT
SDC4	GGCCCTGAAGTTGTCCATCCCT	CATCCTCACTCTCTTCAACGGGTG
SDHA_1	TGGGAACAAGAGGGCATCTG	CCACCACTGCATCAAATTCATG
SDHA _2	TGTTGATGGGAACAAGAGGGCA	GCCTACCACCACTGCATCAAAT
SELL	CAGGCAAATGGAACGATGACGC	ACCCCACATCACAGTTGCAGGT
SERPINB4	ACCAGTGTGGAATCTACTGATTTTGCA	TCGTATCATTGCCAATAGTCCCATCAG
SERPINB5	CTCACTGAAACTAATCAAGCGGCTC	CCTTTGCATACGGTCTCTTCGT
SERPINB13	TGTGCTTCTGCCCAACGACATC	ACCGTCCTCCACCTCAAACCG
SH2D1A	AGTCCTCAGCTAGAAGTACACAAGGT	TGCATTTGTAGCTCACCGAACTGT
SKAP1	TTTGGATCGGAGTGGCAGAAGC	GCCATCCGTACACCGTAGCCC
SMAD1	CAAGAATTTGCTCAGTTATTGGCACAGT	TGGCGGTGGTATTCTGCTCCC
SMAD2	GCTTGAGAAAGCCATCACCACTC	CTCAGTCCCCAAATTTCAGAGCA
SOCS3	AGCCCCAAGGACGGAGACTTC	CGGGAAACTTGCTGTGGGTGAC
SOX2	TGAACCAGCGCATGGACAGTTA	CATCATGCTGTAGCTGCCGTT
SPO11	ACAGAGCAACACTTATGCAACCAAAAG	ACTCCTCCTTGACACTTTTAACATGCA

ST8SIA1	TGTTGGCTCTACATCTTCCCCGT	GGTCGCAGCAGTCTTCCATTTGT
STAG3	TGACAGGGACTCAAACCATACCTCA	TGTTTTCGGTGGTCGTTTTGCTG
STAT1	TGATCTCCAACGTCAGCCAGC	GCCAACTCAGCACTTCTGAAAGC
STAT2	CATTGGAGGGCGCGGGGACT	TCGAATGTCCACAGGCAGGAGG
STAT3	ATGCGGCCAGCAAAGAATCA	AGCGGCTATACTGCTGGTCAAT
STAT4	GGAAATTCGGCATCTGTTGGCC	TTCTCTTTGGAAACACGACCTAACTGT
STAT5A	TGGCAGTGGTTTGACGGGGT	GTCGGGCTTGTTGATGAGCAGG
STAT5B	ACTGAAGATCAAGCTGGGGCA	ACAATATATGGCGGATGCAGCG
STAT6	GGCCACTTTCAGACAAATACTTCAAGGA	TGCAGCCTCCGCAAGCCT
SUZ12	TCATCGCCAACCTGGATTTGCT	ATGTTCTTTGCTGTTCTACTTCCCCAT
SYCP1	CCACCAGCTTCTCATCTTTGTGTCA	AGCAATTACAGCCCAACGGTCC
SYCP3	ACCAAGGCTTCTCTCAAAACTAGTAACC	ATCCCACTGCTGAAACAAAGTCAGA
SYK	TCTTTTTCGGCAACATCACCCG	GCGCAGCAAATAAAGCCCATCA
TBX3	CTGGAGGCTAAAGAACTTTGGGATCA	ATCCAGCCCAGAACATCTCACTTTAAAT
TBX21	ACGCTTCCAACACGCATATCTTTACT	GTTCTCCCGGAATCCTTTGGCA
TCF3	CCTGTTTGAAACGGCGAGAAGA	TGGGGAGCTGAAAGCACCAT
TCF7L1	ACCGTATTACCCACTCTCTCCCG	ATCGAGGCGTTCATGGCGAG
TGFB1	AGTTGTGCGGCAGTGGTTGA	CTTGCAGTGTGTTATCCCTGCT
THAP11	CCAAAGGACGCTGAGTTGCGG	CGTACCGTGTAGGTCTTGCGG
THBS4	AGTTCAGCCACCATCTTCGGTCT	AACCACCAAATGCACCTTCCCA
TIMP1	AGATCCAGCGCCCAGAGAGA	AGCAACAACAGGATGCCAGAAG
TLR7	TGCTCTCTTCAACCAGACCTCTACA	AGTTTTAGGAAACCATCTAGCCCCAAG
TNFRSF4	ACACCTACCCCAGCAACGACC	CACGGCTTGGAGCTGACCAC
TNFRSF8	AGCTCCACCTGTGCTACCCG	CGTTGAGCTCCTCCTGGGTCTG
TNFSF10	TGCAGTCTCTCTGTGTGGCT	GCCACTTTTGGAGTACTTGTCCTG
TNFSF11	TGGTGGATGGCTCATGGTTAGATCTG	CAAGAGGACAGACTCACTTTATGGGAAC
TNFSF13B	TGCAGGGCCACCACGCG	TGCTGTTCTGACTGGAGTTGCC
Tox	TGAGCATGACAGAGCCGAGCC	CAGCGAGTGGTCTGGGAGGG
TP53	ACCATGAGCGCTGCTCAGATA	CCACACGCAAATTTCCTTCCAC
TP63	CGGAGGTGGTGAAGCGGTGC	GCACACTCTGTCTTCCTGTGATGG
TP73	AGCTCGGGAGGGACTTCAAC	AGGGTCATCCACATACTGCGA
TRAF2	ACCGGTACTGCTCCTTCTGC	TCGTGAACACAGGCAGCACA
TRAF3IP3	TGACCACCTCTCCTCACAGGCT	TTGGTTTGCTGACTGGCATCGT
TRIM28	CCCCACAGGAGTTTGCCCAG	GCACAGCAGAGAACTTGGTGTCA
TRIP13	GAGTCGCCAACGGTCCACGT	AAGGTTCATCAAACTCAGTCCATGTGT
TRRAP	GTCCACGCTGATGTTGGAGCA	AGGGAGTAAAGCTCCGCAAGGG
TTR	AGTCTGGAGAGCTGCATGGGC	CGGAGTCGTTGGCTGTGAATACC
TWIST1	TCCATGTCCGCGTCCCACTA	AGCTCCATCCTGGTGTACCTT
VEGFC	GCCAACCTCAACTCAAGGACAGA	CCCCACATCTATACACACCTCCCG
WIF1	GAATTCCTGTCCTTGCGCTCCC	CTGCCACCCCATCCTGTTTTCC
DIABLO	TGCAGTTGGTCTTTCAGAGATGGC	AGCTTGGTTTCTGCTTTCCGGG
XTP6	AGAGTGGAGGCTGGAAGGATGG	TCAGCACAAGGCAAGGATGCTC
YWHAZ	TCCCCAATGCTTCACAAGCAGA	TCTTGTCATCACCAGCGGCAA
ZAP70	ACCCGAATGCATCAACTTCCGC	CTTGCCCTGCTCGATGAAGGC
ZBTB16	CCTGGATAGTTTGCGGCTGAGAA	ATGGGTCTGCCTGTGTGTCTCC
ZFX	GTTGAACTGCTTGATCAGAACAGCAG	TCGGCATGAAGGTTTTGATTTCATTGTC

Supplementary Table 1. Primers used for RT-PCR experiments.

Gene Name	Putative biological role and importance in cancer/CTCL.
Part A. Genes upregulated in Cluster 1 poor prognosis patients.	
IL-26	IL-26 is one of the cytokines produced by the Th17 cells[1].
IL-21 and IL21R	IL-21 is one of the cytokines produced by the Th17 cells[1]. Autocrine IL-21 stimulation was shown to be involved in the maintenance of constitutive STAT3 activation in SS[2].
IL-17A and IL-17F	Both cytokines are typically produced by the Th17 cells[1]. As addressed in the discussion, there remains an uncertainty whether IL-17 signaling plays an important role in CTCL or whether it is seen as a secondary epiphenomenon. Aberrant constitutive activation of JAK/STAT signaling has been documented in CTCL[3]. This constitutive activation of signaling was shown to result in IL-17 production[3].
IL-22	IL-22 was initially thought to be produced by Th17 cells. Later it was shown that IL-22 production can also occur in an unique Th22 subset of cells that lack the ability to produce IL-17 and interferon-γ[4]. IL-22 was proposed to be a dominant cytokine in the tumor microenvironment of CTCL lesional skin[5].
IL2RA	IL-2Rα chain constitutes a part of the high affinity three chain IL-2 receptor. IL-2Rα expression is restricted to T cells that recently encountered an antigen, while in healthy individuals the majority (i.e. >95%) of peripheral T cells are IL-2Rα negative. IL-2Rα was shown to be expressed in patients rejecting allografts, in autoreactive T cells from patients with autoimmune conditions[6]. CTCL malignant cells were shown to constitutively express this protein, which is driven by STAT3 activation[7]. In MF skin and SS circulating T cells IL-2Rα is upregulated in up to 50% of cases. Interleukin-2 diphtheria toxin fusion protein (denileukin diftitox) is designed to target IL-2 receptor in CTCL[6].
ITK	ITK (Interleukin-2 inducible T-cell kinase) is a member of the Tec kinase family of non-receptor tyrosine kinases. It plays an important role in T-cell signaling. Together with other T-cell specific tyrosine kinases (e.g. LCK and ZAP-70) ITK amplifies signals transmitted via a T-cell receptor cascade. Abnormal activity of this cascade was shown to lead to autoimmune disorders and inflammation. ITK-SYK translocations were observed in Peripheral T-cell lymphomas (PTCL)[8].
LCK	LCK (lymphocyte-specific protein tyrosine kinase) is a member of the Src family of protein tyrosine kinases and plays an important role to amplify signals transmitted via a T-cell receptor cascade. Previous cytogenetic studies demonstrated that LCK gene can fuse together with the TCRβ subunit to produce a fusion protein in T-cell acute lymphoblastic leukemia via t(1;7)(p34;q34) translocation. This gene is overexpressed in PTCL. LCK was shown to be able to identify SS from normal control samples[9].
FYB	FYB is an adaptor protein involved in T cell signal transduction pathways. This gene is expressed in T cells, myeloid cells and is believed to promote positive regulation of T cell activation, integrin-mediated adhesion and IL-2 production[9].
GNLY	Granulysin is a cytolytic granule protein, which is expressed in cytotoxic T cells and NK cells. Granulysin is implicated in a variety of diseases including infection, cancer, transplantation, autoimmunity and drug eruptions. Cytolytic proteins including granzyme B and perforin were previously documented to play critical pathophysiological roles in NK/T cell lymphomas. Increased levels of this gene were detected in NK/T cell lymphomas and anaplastic large cell lymphomas (ALCL) cases[10].
GTSF1	GTSF1 (Gametocyte Specific Factor 1) is a cancer testis antigen ectopically expressed in CTCL[11] and was reported to be a part of a molecular signature that is specific to this cancer[12].
SYCP1	SYCP1 (Synaptonemal complex protein 1) is a cancer testis antigen ectopically expressed in CTCL[11]. The normal function of this gene is to regulate crossing over in meiosis[11].

POU2AF1	POU2AF1 is a B cell-specific transcriptional factor. This gene is essential for B cell maturation and germinal center formation[13]. Previous research demonstrated ectopic expression of B cell specific genes in CTCL, as in the case of BLK gene[14].
CHD1	CHD1 (Chromodomain-helicase-DNA-binding protein 1) is an embryonic gene, whose activity is required to maintain open chromatin of pluripotent mouse embryonic stem cells[15].
TOX	TOX is a transcription factor, which is essential for early development of $CD4^+$ T cells and is normally not expressed in mature $CD4^+$ T cells. Previous studies demonstrated that the $CD4^+$ T cells in CTCL ectopically express TOX, which causes the proliferation/apoptosis balance to shift toward proliferation by suppressing the transcription of several tumor suppressors. It was previously proposed that targeting TOX activity may be a promising treatment strategy for CTCL. TOX expression was independently found by two separate laboratories to be a robust diagnostic and prognostic marker for this cancer[16,17].
TCF3	TCF3 (Transcription factor 3) is a member of Tcf/Lef transcription factor family. LEF1 and TCF1 are required for transactivation of Wnt signaling genes, while TCF3 functions predominantly as a transcriptional repressor. Notably, TCF3 is expressed in different types of stem cells including embryonic and hair follicle stem cells. TCF3 promotes differentiation of embryonic stem cells by counteracting Wnt-mediated maintenance signals[18]. TCF3 is believed to be an important negative regulator of embryonic stem cell self-renewal. Chromosomal translocation t(1;19)(q23;p13.3) which leads to a production of the TCF3-PBX1 (E2A-PBX1) fusion protein was observed in Acute Lymphoblastic Leukemia. Recurrent mutations in TCF3 that promote PI3-kinase signaling were documented in human Burkitt's lymphoma samples[19]. While this gene is upregulated in CTCL it is not known if it is also mutated.
LEF1	LEF1 (Lymphoid enhancer-binding factor 1) is a downstream member of the Wnt/β-catenin signaling pathway. Increased LEF1 expression has been reported as a poor prognostic marker in various hematologic malignancies[20].
NFKB1	NFKB1 is a master transcription factor that plays a major role in inflammatory and immune responses. Activation of the NFκB signaling pathway plays a critical role in the development and progression of many types of cancer. NFKB1 was shown to be overexpressed in MF patients and may correlate with poor disease outcome[21].
JUNB	c-JUN and c-FOS were initially identified as viral oncoproteins and their roles in tumorigenesis are well established. Previous studies documented gain in copy numbers of JUNB in 54% of c-ALCL and 26% of SS/MF patients with strong nuclear expression of JUNB protein in these cancers. Dysregulation of AP-1 expression in CTCL was also shown to be associated with genomic amplification of JUNB in this cancer. JUNB is an important transcriptional regulator of IL-4 expression and is associated with the Th2 phenotype in the advanced CTCL[22].
CCL18	CC chemokine ligand (CCL) 18 is produced by monocytes and dendritic cells and was shown to act as a potent chemoattractant for T and B cells. CCL18 expression is upregulated in atopic dermatitis and bullous pemphigoid. The CTCL lesional skin was shown to express elevated levels of CCL18 mRNA in comparison to normal skin. Further studies showed that dermal macrophages and dendritic cells in CTCL skin were responsible for CCL18 production. Serum levels of this protein were elevated in CTCL and correlate with the types of skin lesions. Patients with high serum levels of CCL18 had more aggressive disease course than patients with low CCL18 levels[23].
CCR4	CC chemokine receptor 4 (CCR4) is highly expressed in SS and MF skin in all CTCL subtypes. Expression of CCR4 is limited in non-malignant cells as it is absent in neutrophils, monocytes, B cells and naïve T cells. It is also expressed in fewer than half of all memory T cells. Based on these findings Mogamulizumab, humanized anti-CCR4

	antibody, was proposed as an attractive therapeutic target for CTCL and is currently being tested in patients[24].
STAT5A	Upregulation in STAT5 signaling occurs in early CTCL stages. A growing body of experimental evidence suggests that this gene is important for expression of anti-apoptotic proteins (bcl-2 and bcl-x), cell cycle genes (Cyclin D and c-myc) and oncogenic miR-155 microRNA, all working in concert to promote cancerogenesis[25,26].
SH2D1A	SH2D1A (SH2 domain-containing protein 1A) was shown to be associated with X-linked lymphoproliferative disease[27].
TFRC (CD71)	Iron is transported in serum by transferrin and enters the cell via the transferrin receptor (TFRC). In replicating cells iron is required for DNA synthesis and cytochrome function. To meet this need, as proliferating cells exhaust available intracellular iron, they increase their surface expression of TFRC. Activated T cells express surface receptors including CD25 (the IL-2 receptor) and TFRC/CD71. Previous reports document upregulation of T cell activation markers including TFRC/CD71 in CTCL[28].
MXI1	MXI1 protein is a basic helix-loop-helix, leucine zipper transcriptional factor that can dimerize with Max protein and bind to specific DNA sequences and suppresses the transcription of genes that are typically transactivated by a c-Myc/Max dimer[29].
AHI1	Abelson Helper Integration site 1 (AHI1) and downstream signaling members, (e.g. CDKN1C), were suggested to play an important role in CTCL carcinogenesis[19,91]. AHI1 is typically activated by provirus insertional mutagenesis in various murine leukemias and lymphomas. Overexpression of this gene was demonstrated in Hut102 and Hut78 CTCL cell lines. One of the putative functions of AHI1 is to suppress CDKN1C[30,31].
IRF4	The IFN regulatory factor 4 gene, also known as multiple myeloma antigen 1 (MUM1), is normally expressed in plasma cells, melanocytes, subset of B cells, and in activated T cells. It is required for B-cell development, plays an important role in Th2, Th17 T-cell differentiation and T-cell cytotoxic function. In some multiple myeloma, IRF4 is involved in t(6;14)(p25;q32) reciprocal translocation, which leads to the juxtaposition of this gene next to the immunoglobulin heavy chain locus. IRF4 rearrangements were previously documented is a subset of diffuse large cell B-cell lymphoma, splenic marginal zone lymphoma, chronic B-cell lymphoid leukemia, transformed MF and ALCL cases. IRF4 locus amplification was observed in a subset of c-ALCL and transformed MF/ SS cases[32].
PLK1	Polo-like kinases belong to the serine/threonine kinase family and is critical for mitosis and DNA integrity. PLK1 is one of the most studied members of this family and was found to be upregulated in a variety of cancers. It is also upregulated in the G2/M phase of mitosis. PLK1 was found to be overexpressed in advanced lesions of CTCL and in several CTCL cell lines including HH, Hut78, MyLa, SeAx and SZ4. Downregulation of this gene results in decreased malignant cell proliferation and viability[33].
NAIP	NLR apoptosis inhibitory protein (NAIP) is homologous to two baculovirus inhibitor of apoptosis proteins (IAP) and is able to suppress apoptosis induced by various signals[34]. Resistance to apoptosis is believed to be one of the cardinal features of CTCL[34].
CCND2	CCND2 (G1/S-specific cyclin-D2) upregulation was documented in a subset of CTCL cases and in a variety of leukemias and lymphomas[35].
T3JAM	T3JAM (TRAF3-interacting JNK-activating modulator) is expressed in bone marrow, spleen and thymus and was shown to promote specific activation of JNK signaling[36].
Part B. Genes upregulated in Cluster 2 favorable prognosis patients.	
LCE2B	LCE2B (Late Cornified Envelope Protein 2B) is one of at least 20 genes that are expressed during epidermal differentiation. This gene was found to be expressed in normal and psoriatic skin, but not in cultured keratinocytes or in other tested cell types or tissues[37].

CST6	CTS6 (Cystatin 6), also known as cystatin E/M, is a cysteine protease inhibitor that is downregulated in breast, cervical, glioma, prostate and gastric cancers. Loss of CST6 expression is attributed to promoter hypermethylation. Also, CST6 has been shown to be important for skin differentiation. This gene is proposed to act as a tumor suppressor gene by controlling the activity of a known oncogene, Legumain (LGMN)[38].
LOR	Loricrin is a major protein component of the cornified cell envelope that is responsible for protective barrier function of the stratum corneum. This gene is expressed in terminally differentiated keratinocytes. Mutations in this gene have been reported in Vohwinkel's keratoderma and progressive symmetric erythrokeratoderma[39].
LTBP4	The extracellular matrix protein LTBP4 (latent transforming growth factor β-binding protein 4) belongs to the fibrillin/LTBP family of glycoproteins. These proteins can covalently bind to TGFβ and play an important role in promoting the folding and secretion of this protein. Dysregulated expression of LTBP isoforms was shown to be associated with epithelial neoplasms. Specifically, LTBP1 is downregulated in neoplasms of the liver, ovaries and neuroendocrine tumors of the digestive system. LTBP2 is downregulated in esophageal squamous cell carcinoma and nasopharyngeal carcinomas. LTBP4 is downregulated in breast adenocarcinomas. Promoter hypermethylation was documented to be the mechanism of downregulation for this gene[40]
BCL7A	BCL7A (B-cell CLL/lymphoma 7A) putative tumor suppressor gene that was previously suggested by us and others to play an important role in CTCL carcinogenesis and progression[20,36,37]. It is often down-regulated in CTCL patients when compared to benign skin conditions. Several mechanisms for this loss of expression have been demonstrated, where BCL7A and the corresponding 12q24.31 region of the chromosome were lost in 56% of patients with CTCL. In another study, 48% of patients exhibited BCL7A promoter hypermethylation. Such promoter hypermethylation was preferentially observed in patients with aggressive CTCL. This gene may be an important prognostic marker in patients with early-stage disease[41-43].
CDKN1C	CDKN1C (Cyclin-Dependent Kinase Inhibitor 1C) belongs to the Cip/Kip family of cyclin-dependent kinase inhibitors, which negatively regulate cell cycle progression by inhibiting G1 cyclin-dependent kinases. Mutations in this gene were identified in patients with Beckwith-Wiedemann syndrome, which is characterized by an over-growth phenotype and an association with several cancers. Hence, loss-of-function of CDKN1C promotes cell proliferation giving rise to an over-growth phenotype[44]. Our previous work suggests that this gene is a downstream target of AHI1 oncogene and its loss may play an important role in CTCL carcinogenesis[31].
PSORS1C2	PSORS1C2 is a poorly characterized psoriasis susceptibility gene[45].
WIF1	WIF1 (WNT inhibitory factor 1) is a WNT/β-catenin signaling inhibitor and was previously shown to be downregulated in salivary gland carcinomas[46], acute lymphoblastic leukemias[47] and acute myeloid leukemias[48]. Our studies[49,50] for the first time suggest that loss of this gene may also be important for CTCL carcinogenesis.
DLEU1	DLEU1 (Deleted in Lymphocytic Leukemia 1) is a long non-coding RNA putative tumor suppressor gene and is frequently deleted in B-cell chronic lymphocytic leukemia[51] and may have tumor suppressing properties in CTCL[52].
IL-18	IL-18 proinflammatory cytokine is known to induce expression of interferon-γ and promote Th1 immune responses, both of which are associated with disease clearance[53].
IL1F7 (IL-37)	IL1F7 is a target gene upregulated by IL-18 signaling and acts as an inhibitor of innate immunity[54].
miR-205	miR-205 microRNA was documented to act as putative tumor suppressor gene that targets E2F1 in melanoma and other cancers and has the ability to distinguish CTCL from other benign entities[55].

Part C. Genes upregulated in Cluster 3 intermediate prognosis patients.	
FOSL1	FOSL1 (Fos-Like Antigen 1), also known as FRA1, forms a part of the AP-1 complex. Gain of function of this oncogene has been linked to the enhanced migration and invasion of colorectal breast, lung, bladder, head and neck, thyroid and brain carcinomas. FOSL1 expression is induced by RAS-ERK and Wnt/β-catenin pathways[56].
SERPINB4	SERPINB4, also known as squamous cell carcinoma antigen 2 (SCCA2), is a member of the ovalbumin family of serine proteinase inhibitors. It was originally isolated from metastatic cervical squamous cell carcinoma. SERPINB4 is expressed primarily in malignant cells and correlates with more aggressive tumors[57].
EVA1	EVA1 (Epithelial V-like antigen 1), also known as MPZL2 is expressed in the thymus early in embryogenesis and subsequently is downregulated during thymocyte developmental progression. It is believed to contribute to the earliest phases of thymus organogenesis[58].
MMP12	MMP12 (matrix metalloproteinase-12) also known as a macrophage metalloelastase or macrophage elastase is an enzyme involved in the breakdown of extracellular matrix in normal physiological and pathological processes. MMP12 may play a role in aneurysm formation, in the development of emphysema and was recently shown to promote migration and invasion in nasopharyngeal carcinoma[59].
SELE	SELE (E-selectin), also known as CD62 antigen-like family member E (CD62E), endothelial-leukocyte adhesion molecule 1 (ELAM-1), or leukocyte-endothelial cell adhesion molecule 2 (LECAM2), is a cell adhesion molecule expressed by activated endothelial cells. It plays an important role in inflammation. Previous studies that analyzed E-selectin staining in blood vessels showed differences between various disease groups and healthy controls, with the highest percentages being observed in CTCL patients[60].
CCL26	CC chemokine CCL26, also known as eotaxin-3, is a potent chemoattractant and was shown to correlate with the clinical itch burden in CTLC patients[61].
PLS3	PLS3 (Plastin-3) is an actin-binding protein, which is not normally expressed in T cells. It was shown that the promoter for this gene is demethylated in cancer. The normal function is to regulate actin structure elongation. This gene is associated with SS cell survival and migration and was shown to be overexpressed in SS cells by many studies[62].

Supplementary Table 2. Detailed description of the 52 genes that fit into the three signature pattern expression model and their putative functions in CTCL and other malignancies.

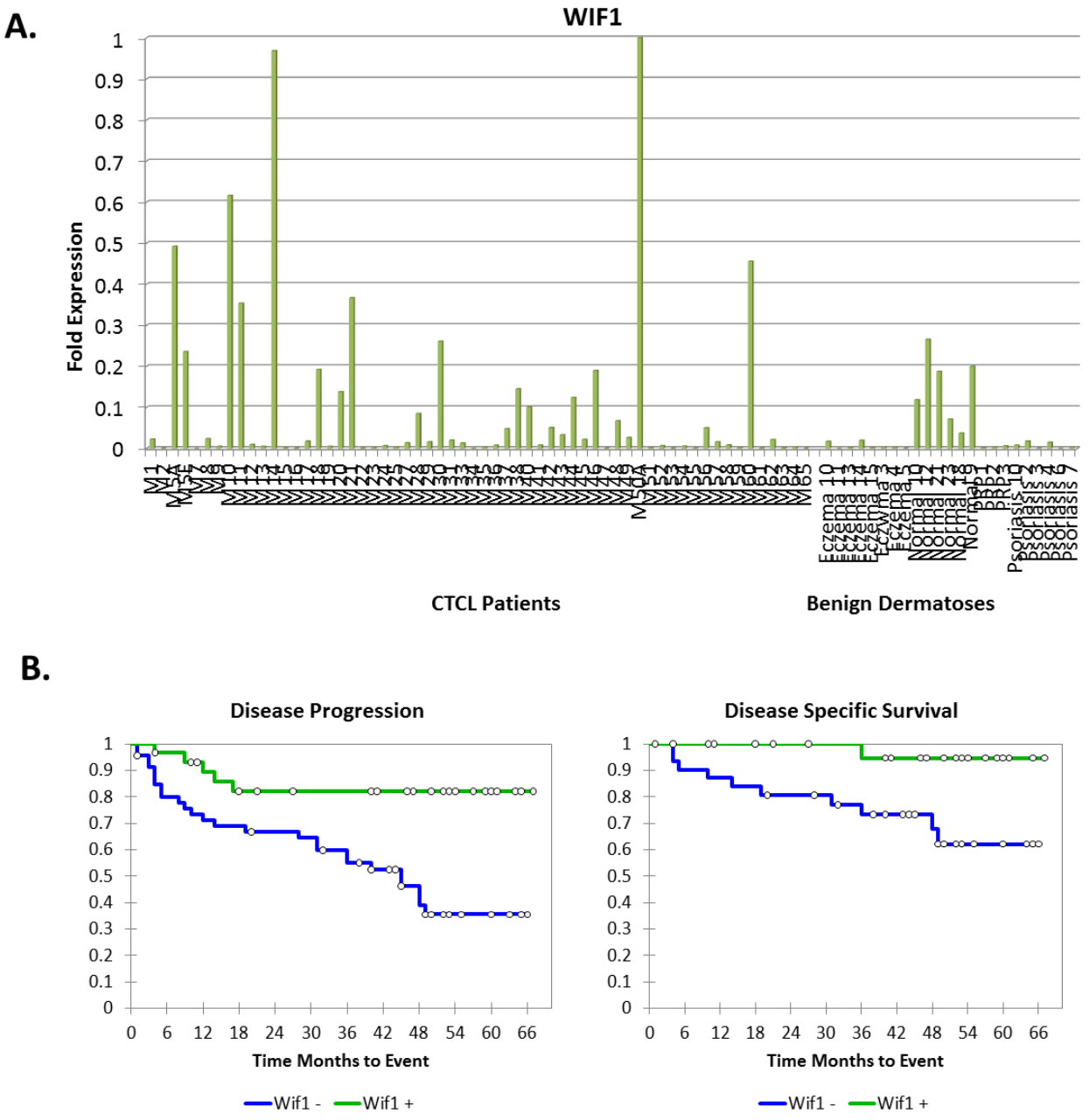
Analysis of Maximum Likelihood Estimates (Cox Model)	
Patient Characteristics	Odds Ratio for Progression (p value)
Age	
< 40	1.77 (p=0.51)
40-59	1.48 (p=0.50)
≥ 60	1 (reference)
Sex	
Male	1.65 (p=0.25)
Female	1 (reference)
Clinical Stage at the Time of Diagnosis	
Stage I	1 (reference)
Stage II	4.7 (p=0.005)
Stage ≥ III	12.0 (p<0.0001)

Supplementary Table 3. Multivariate analysis of patient characteristics that are associated with clinical disease progression.

Gene Name	Putative biological role and importance in cancer/CTCL.
Part A. Genes preferentially expressed in CTCL patients.	
CCL26	Please see Supplementary Table 2C for more details.
CCL18	Please see Supplementary Table 2A for more details.
FYB	Please see Supplementary Table 2A for more details.
IL2RA	Please see Supplementary Table 2A for more details.
LEF1	Please see Supplementary Table 2A for more details.
LCK	Please see Supplementary Table 2A for more details.
ITK	Please see Supplementary Table 2A for more details.
TOX	Please see Supplementary Table 2A for more details.
CCR4	Please see Supplementary Table 2A for more details.
GNLY	Please see Supplementary Table 2A for more details.
MMP12	Please see Supplementary Table 2C for more details.
T3JAM	Please see Supplementary Table 2A for more details.
IL1F7	Please see Supplementary Table 2B for more details.
IL-22	Please see Supplementary Table 2A for more details.
IL-26	Please see Supplementary Table 2A for more details.
STAT5A	Please see Supplementary Table 2A for more details.
SYCP1	Please see Supplementary Table 2A for more details.
GTSF1	Please see Supplementary Table 2A for more details.

cTAGE1	This cancer testis antigen was documented to be ectopically expressed in CTCL. cTAGE1 (Cutaneous T-Cell Lymphoma-Associated Antigen 1) is robustly expressed in the majority of CTCL patients and patient-derived cell lines[11].
CDO1	CDO1 (cysteine dioxygenase) was shown to be consistently overexpressed in SS patients and is the rate-limiting enzyme in the synthesis of taurine, an important semi-essential amino acid. CDO1 is not usually expressed in peripheral blood. The expression of this gene was documented in liver and brain. The CDO1 promoter, is believed to be under the regulation of c-myb, which is consistently overexpressed in PBMCs from SS, but not MF patients[63].
THAP11	THAP11 (Thanatos-associated protein 11), also known as Ronin, is essential for the self-renewal of embryonic stem (ES) cells. This gene contributes to ES cell pluripotency by regulating the transcription of genes involved in the metabolic processes that sustain the growth of self-renewing ES cells[64].
STAT4	STAT4 appears to be overexpressed in the early stages of CTCL when compared to benign skin diseases[52]. The expression of STAT4 is required for Th1 differentiation. The expression of this gene is lost in advanced CTCL with concomitant shift towards the Th2 phenotype[26,65,66]. Loss of STAT4 expression appears to be a robust and reliable diagnostic marker for SS[66].
Part B. Genes preferentially expressed in normal skin and in patients with benign inflammatory dermatoses.	
WIF1	Please see Supplementary Table 2B for more details.
BCL7A	Please see Supplementary Table 2B for more details.
PSORS1C2	Please see Supplementary Table 2B for more details.
DMAP1	DMAP1 participates in DNA repair by directly interacting with PCNA. DMAP1-depleted cells in p53-deficient background demonstrate chromosomal instability and tumor formation in mice. Recent reports indicate that DMAP1 acts as a tumor suppressor by maintaining chromosomal integrity[67].
SERPINB13	SERPINB13 is an inhibitor of lysosomal cathepsin enzymes K and L and was shown to be downregulated in the head and neck cancers. Cathepsin K promotes cancer cell invasion via degradation of the extracellular matrix[68].

Supplementary Table 4. Description of genes that are able to distinguish CTCL from benign dermatoses and their putative roles in CTCL and other malignancies.



Supplementary Figure 1. A. WIF1 (Wnt Inhibitory Factor 1) gene is expressed in normal skin and CTCL lesional skin in a subset of patients. **B.** Correlation of WIF1 expression in CTCL patients with disease progression. Kaplan-Meier analysis documents that loss of WIF1 expression is associated with poor CTCL disease progression (left panel, p=0.002). Disease progression is defined as a progression to a higher clinical stage and/or death. Loss of WIF1 is also associated with poor cancer-specific survival (right panel, p=0.012).

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