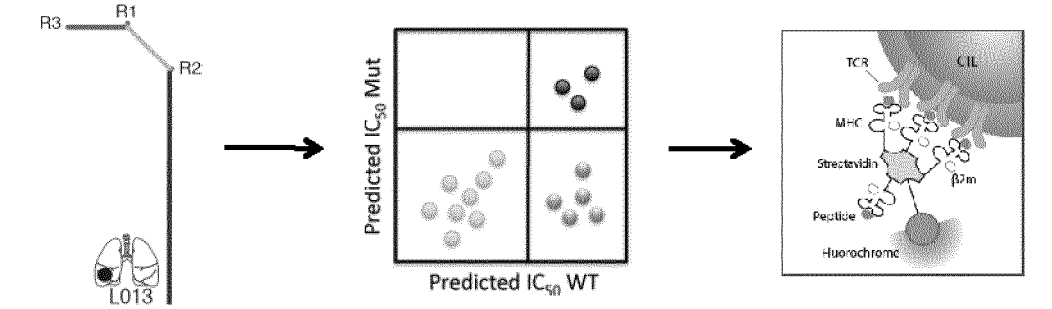
FIGURE 1



Mutational heterogenity in NSCLC

Predicted Neo-antigenic landscape in NSCLC

Identification of neo-antigen reactive TILs by soluble peptide-HLA tetramer technology

FIGURE 2

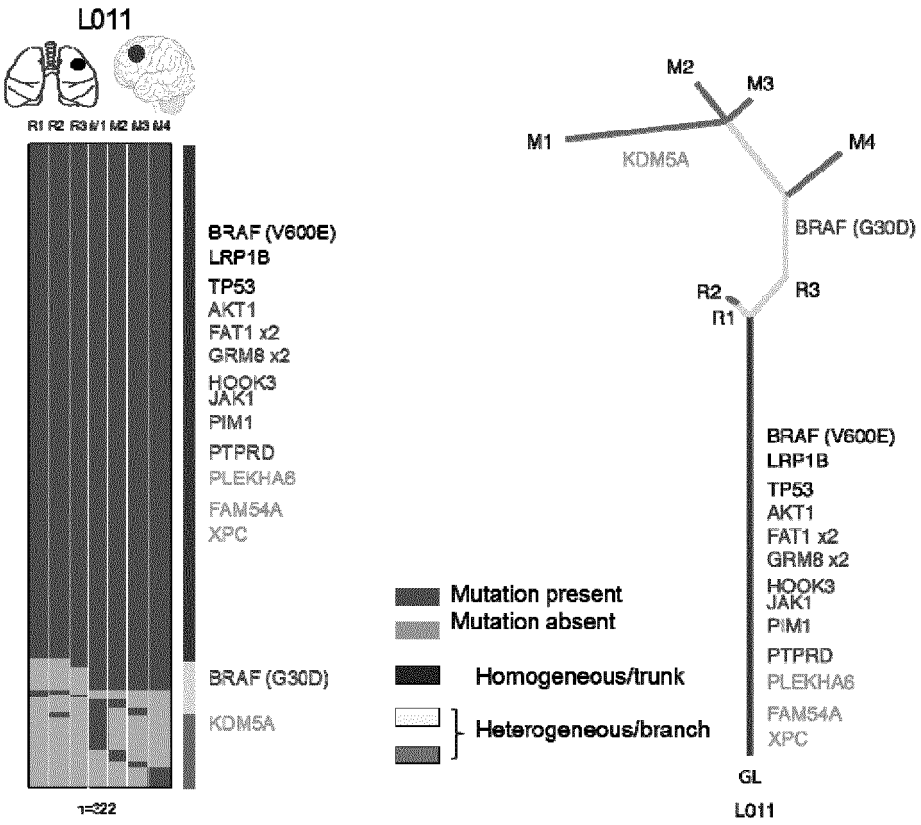
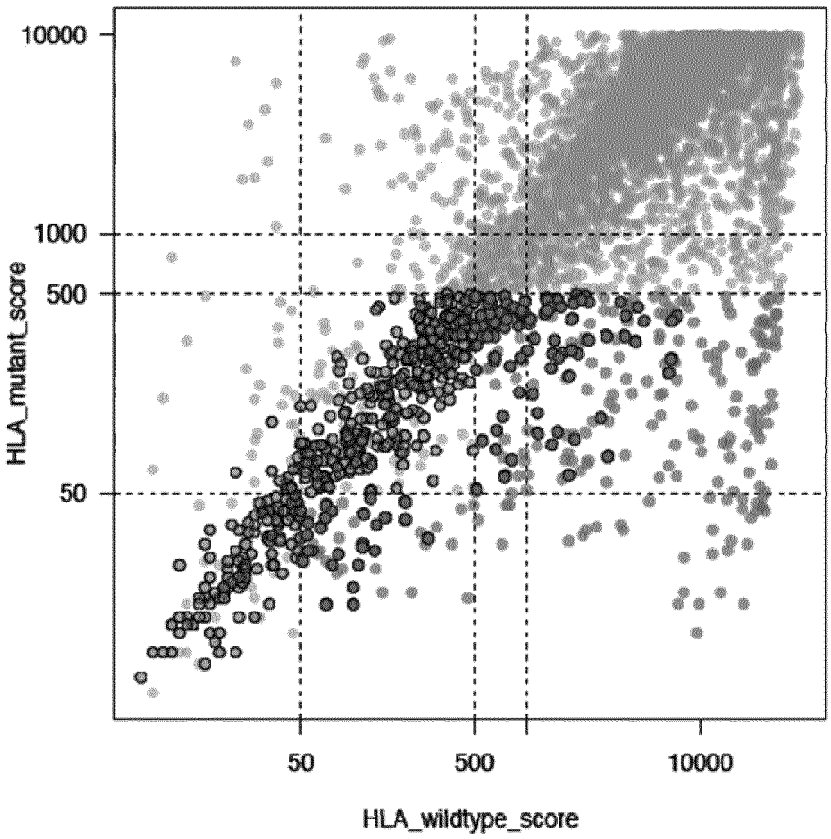

L011
R1 R2 R3 M1 M2 M3 M4
BRAF (V600E)
LRP1B
TP53
AKT1
FAT1 x2
GRM8 x2
HOOK3
JAK1
PIM1
PTPRD
PLEKHA6
FAM54A
XPC
BRAF (G30D)
KDM5A
n=22
Mutation present
Mutation absent
Homogeneous/trunk
Heterogeneous/branch
M2
M3
M1
KDM5A
M4
BRAF (G30D)
R2
R3
R1
BRAF (V600E)
LRP1B
TP53
AKT1
FAT1 x2
GRM8 x2
HOOK3
JAK1
PIM1
PTPRD
PLEKHA6
FAM54A
XPC
GL
L011

FIGURE 3



Mutations identification by Exome seq and RNAseq

Predicted binding of patient's mutations to patient's HLA

Selection of 200 peptides with high predicted HLA binding affinity

Tetramer generation and identification of neoantigen-specific T cells

FIGURE 4

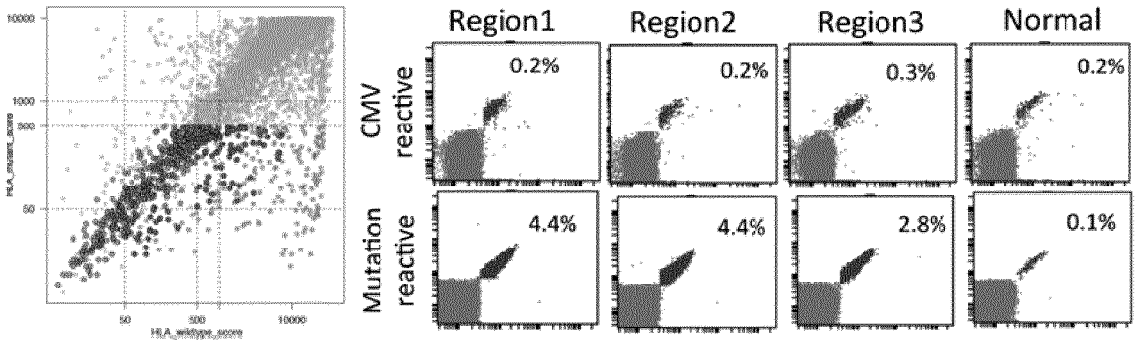
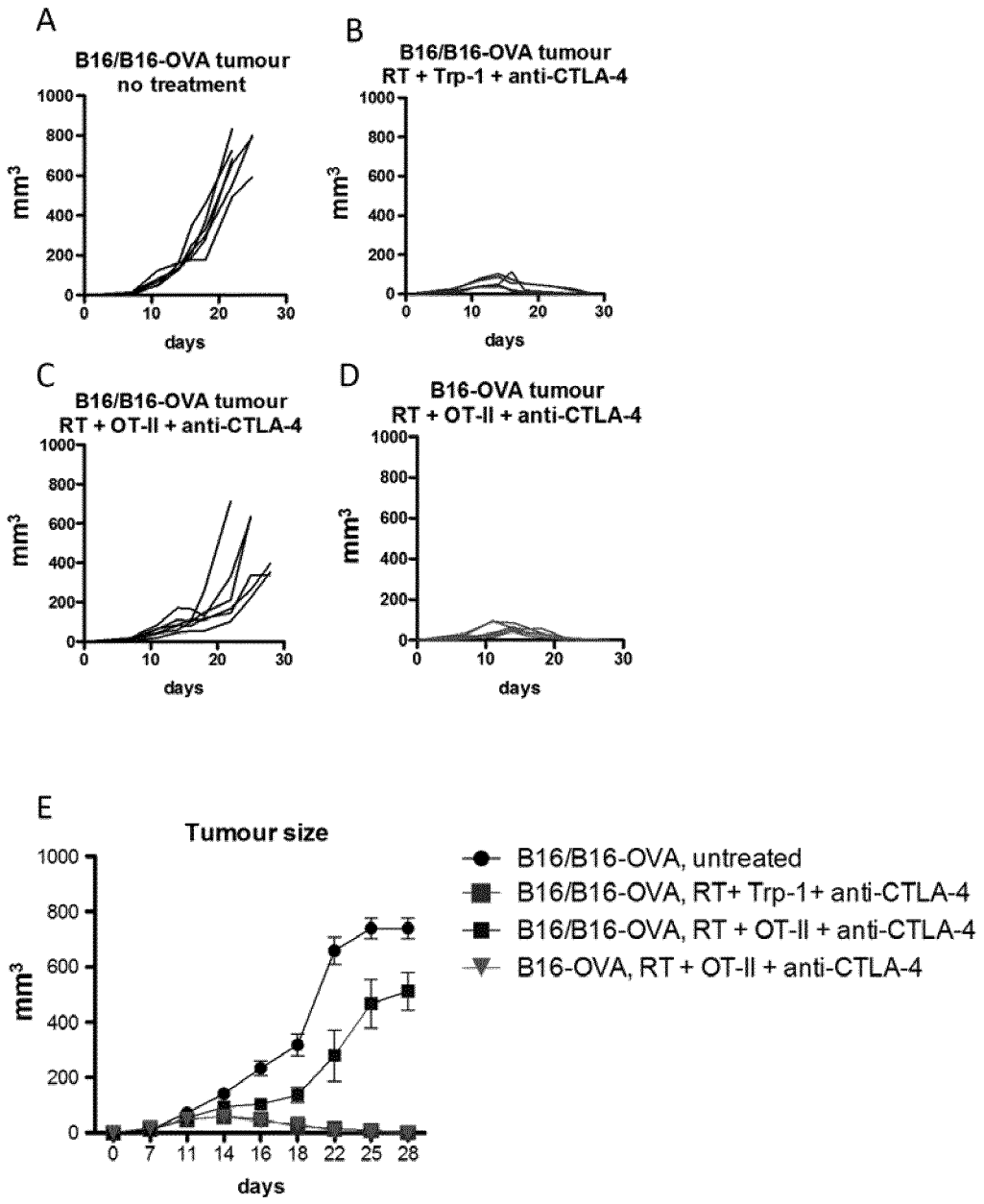

10000
1000
500
50
50
500
10000
Region1
Region2
Region3
Normal
CMV reactive
Mutation reactive
0.2%
0.2%
0.3%
0.2%
4.4%
4.4%
2.8%
0.1%

FIGURE 5


A
B16/B16-OVA tumour
no treatment
mm3
days
B
B16/B16-OVA tumour
RT + Trp-1 + anti-CTLA-4
mm3
days
C
B16/B16-OVA tumour
RT + OT-II + anti-CTLA-4
mm3
days
D
B16-OVA tumour
RT + OT-II + anti-CTLA-4
mm3
days
E
Tumour size
mm3
days
B16/B16-OVA, untreated
B16/B16-OVA, RT+ Trp-1+ anti-CTLA-4
B16/B16-OVA, RT + OT-II + anti-CTLA-4
B16-OVA, RT + OT-II + anti-CTLA-4