Supplementary Methods

Inclusion and exclusion criteria

Other relevant inclusion criteria were: patients had to have measurable disease according to RECIST 1.1, compulsory tumor biopsy at baseline and at week 13th for translational purposes, Eastern Cooperative Oncology Group (ECOG) performance status of 0-1 and adequate hepatic, renal, cardiac and hematologic function. Most significant exclusion criteria were four or more previous lines of chemotherapy for the advance disease, previous administration of anti-programmed death-1 (PD.1), anti-programmed death-ligand (PD-L1), anti PD-L2 or anti CTLA-4. Grade 3 or higher immune-related adverse event (pneumonitis, hepatitis, colitis, endocrinopathies) with prior immunotherapy or active autoimmune were also exclusion criteria.

Procedures

General dose-modification rules for sunitinib recommended withhold dose if toxicity grade 3 or 4 occurred until toxicity reduced up to grade ≤ 1, then resume the treatment at the same dose level, it could be considered dose reduction if previous grade 4 toxicity occurred. Nivolumab dose should be delayed if any grade ≥ 2 occurred with the exception of grade 3 skin toxicity, grade 2 fatigue or laboratory abnormalities. More detailed specific rules for dose modifications are described in the protocol.

Even when a 3+3 design was followed, the recommended dose-level cohort should contain at least 10 patients for obtaining a higher safety information since this combination was probed to be toxic in renal carcinoma.[1] Besides, a

de-escalation was foreseen for the same reason and after considering that the optimal biological dose may be lower than de maximum tolerated dose.[2]

The following adverse effects observed during the 28-day observation (from day 15 to 42) were considered DLT, grade 4 thrombocytopenia, grade 3 thrombocytopenia with bleeding, febrile neutropenia, non-hematologic toxicity ≥ grade 3 or inability to complete ≥ 75% of sunitinib treatment or 2 consecutive doses of nivolumab due to study treatment-related toxicity.

For both, phase Ib and II parts, treatment was continued until disease progression (according to RECIST 1.1), unacceptable toxicity, withdrawal of consent, a delay in treatment administration longer than 3 weeks, or by investigator decision in the context of non-compliance with the protocol requirements.

Independent central review was mandatory, so that the centres had to upload anonymously the studies onto a web-based imaging platform for each assessment and it was reviewed by blinded independent central evaluation.

Assessment of adverse events included type, severity (graded by the National Cancer Institute [NCI] Common Terminology for Adverse Events [CTCAE, version 4.0]), timing, seriousness, and relatedness with investigational compounds. The adverse effects were monitored every other week, coincident on the nivolumab administration days.

Laboratory investigations included clinical biochemistry, blood count cells, coagulation test, and urinalysis for proteinuria, thyroid function test and pregnancy tests.

Phase Ib secondary outcomes

Secondary objectives were toxicity profile according to CTCAE version 4.0, overall response rate (ORR) following RECIST 1.1, 6-month PFS, OS and to contribute to the translational studies.

HTG Molecular OBP

Targeted RNA-Seq was used to determine potential prognostic and/or predictive biomarkers. Gene expression levels were determined on pre-treatment FFPE samples, using the HTG Oncology Biomarker Panel (OBP). Median OS (mOS) and PFS at 6-month were taken as grouping criteria for bioinformatic translational analysis. The HTG EdgeSeq Oncology Biomarker Panel (OBP) was chosen to measure the potential predictive value of 2549 human RNA transcripts (https://www.htgmolecular.com/assays/obp), associated with tumour biology. Only samples with a minimum of 70% of tumour area, were initially considered for transcriptomics analyses, whereas samples with less than 70% tumour or greater than 20% necrotic tissue underwent macro-dissection. Of 65 tumour samples collected at baseline in the STS cohort, 28 had enough tissue for HTG EdgeSeq direct-transcriptomic analyses and were used for this assay.

RNA-Seq libraries were synthetize with the HTG EdgeSeq Chemistry; samples were lysed and permeabilized to expose mRNA, which was hybridized with Nuclease Protection Probes (NPPs). The S1 nuclease was added to the mix, producing a stoichiometric amount of target mRNA/NPP duplexes. S1 nuclease activity was inactivated by heat. Before being included in the HTG EdgeSeq system, samples were randomized to decrease potential biases in the experiment. PCR reactions were performed with hybridized samples and using common adaptors essential for clustering on an Illumina sequencing platform

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and specially designed tags, sharing common sequences that are complementary to both 5'- end and 3'- sequences of the probes. These tags contain a unique barcode that is used for sample identification and multiplexing. Then, the PCR product was clean-up using Agencourt AMPure XP (Beckman Coulter).

The library was quantified by quantitative PCR, with KAPA Library Quantification kit (Roche), according manufacturer´s instructions. All samples and controls were quantified in triplicate and no template control was included in each run. The denaturation of libraries was achieved by adding first 2N NaOH, followed by the addition of 2N HCl. The PhiX was spiked at a 5% (concentration of 12.5 pM). The normalized libraries were sequenced by NGS.

A demultiplexed FASTQ file was retrieved for each sample for data processing. HTG EdgeSeq host software was used to align the FASTQ files to the probe list. Afterwards, the results were parsed and the output obtained as a read counts matrix.

Data filtering and normalization

The baseline performance was evaluated, using negative control probes as quality control, as previously described.[3] More precisely, the mean of negative probes for each sample was calculated, and the difference between negative control average and the mean of all negative control probes was obtained (Δmean). Those samples with a Δmean outside the bounds of +/-2SD were excluded from final data analyses.

The trimmed mean of M-values (TMM) method was applied for data normalization, using the EdgeR package from R/Bioconductor, adjusting for the total reads within a sample.[4] Genes with uniformly low expression were

removed from data analyses and those genes with an expression value over all negative control probes (maximum value of 42.21) in at least 3 samples were selected.

Differential gene expression

For bioinformatics data analyses, samples were grouped according to phase II primary endpoint (PFS rate at 6-months) or median OS. The better prognosis group included samples with a PFS value higher than 6.0 months, while the worse prognosis group embraced samples with PFS lower than 6.0 months. Likewise, better prognosis group included samples with an OS higher than the median (17.4 months), while the worse prognosis group comprised those cases with an OS lower than 17.4 months.

Differential gene expression was evaluated considering all the 2549 transcripts included in the OBP assay. However, to better define the role of immune-system in trial patient outcome, differential gene expression was also performed taking into account only the genes focused on tumour/immune interaction that are included in the OBP assay. Accordingly, of the 2549 genes included in the OBP, 732 genes were selected and used in this analyses. These genes were selected based on the HTG EdgeSeq Precision Immuno-Oncology (PIO) Panel, which measures the immune response in both tumor and its surrounding microenvironment. Of the 1392 genes that compose the PIO panel, 732 are present in the OBP assay.

A negative binomial generalized log-linear model was applied to evaluate differential gene expression, using the EdgeR package and implementing the method proposed by Robinson and Smyth.[5] Benjamini & Hochberg correction for multiple comparisons was applied and a p-value threshold of 0.05 was set

for significance.[6] Fold change values were obtained, along with p-values and adjusted p-values for all the genes evaluated. None of the genes analysed remained significant after Benjamini-Hochberg multiple comparisons correction. Batch effect was taken into account by adding batch information to the model as a co-variable. For data visualization and later analyses, normalized log-cpm values were obtained and variability due to batch effect was removed using removeBatchEffect method implemented in limma R package.

Hierarchical clustering was performed using normalized expression data from genes significantly different according to the OS, for this purpose, Euclidean distance between samples and genes was calculated and samples were then clustered using complete-linkage clustering method scaled by gene.[7]

Samples were categorized according to the groups obtained by this clusterization and those overexpressed and infra-expressed genes characterizing each group were selected after performing a t-test to evaluate median differences between each group and the remaining groups altogether. Using enrichR tool, available as an R package[8 9], we performed an enrichment analysis of the selected genes in KEGG Human Pathways, obtaining those cell functions enriched in the genes comprising each signature. This analysis was performed for each group independently and considering the genes overexpressed in each group.

All analyses were performed with R/Bioconductor (3.10) running on R version 3.6.0.

ProcartaPlex multiplex immunoassays

The levels of 65 soluble protein targets were determined in plasma samples of 10 patients enrolled in phase Ib part of the trial, using the Human Immune

Monitoring 65-plex ProcartaPlex Panel (Invitrogen, Carlsbad, CA, USA), as an exploratory analysis. Plasma was separated from peripheral blood samples using Ficoll-Paque®. The ProcartaPlex multiplex immunoassay was performed following manufacturers' instructions and using 20µl of plasma. The protein levels were quantified using Luminex technology.

Observed signalling from standard samples (with known protein concentration) was used to obtain the calibration line, then, data was fitted to a linear model and expected protein concentration values were predicted. These values were used as protein concentration data for univariate statistical analyses.

Statistical analysis

Variables following binomial distributions (i.e. proportion of OR) were expressed as frequencies and percentages. In univariate analysis were analysed the following clinic-pathological factors as categorical variables: age (categorised according to the median value), MFI, baseline ECOG or extension, histology group, number of previous lines and previous antiangiogenic therapy. Genes proven to be differentially expressed in the bioinformatics' analysis were also analysed in a univariate analysis. These genes were selected based on its p-value and fold change determined in the bioinformatic analysis. Nevertheless, *PDCD1* (PD-1) and *CD274* (PD-L1) were also analysed, independently of its p-value and fold change. For the translational study, selected genes were categorized using ROC curves for their impact in progression and death. All p-values reported were two-sided, and statistical significance was defined at $p<0.05$. The software package used for statistical analysis was SPSS Statistics (version 26).

Supplementary results

Previous systemic lines

The median of previous systemic lines, before enrolment, were 1 (0-4), 45 of 52 patients (87%) had received at least one previous systemic line and 24 of 52 patients (46%) had received more than one previous systemic line. Previous antiangiogenic lines were reported in 11 of 52 patients (21%).

Biomarkers analysis

Among the 2.549 genes screened at baseline 274 and 326 genes showed prognostic role for PFS or for OS, respectively. *PDGFD* and *IL16* showed to be the most significant predictors for prognosis (table 3). Overexpression at baseline of IL-16 in plasma was also significantly associated with better OS: 20.6 (95% CI 19.2-20.1) vs 8.4 (95% CI 6.2-10.5), p=0.027.

Selection of genes for functional enrichment analysis

Hierarchical clustering of the 102 genes with impact in OS, classified samples in two groups with different outcomes. For KEGG functional enrichment analysis and to evaluate whether the two groups have prognosis value on its own or not, samples were re-categorized according to OS, and the differences in gene expression evaluated. A total of 84 genes showed different mean values in both groups, of which 59 withstood Bonferroni correction (Supplementary Table 2). 70 genes were overexpressed in group 2 and 14 in group 1.

References

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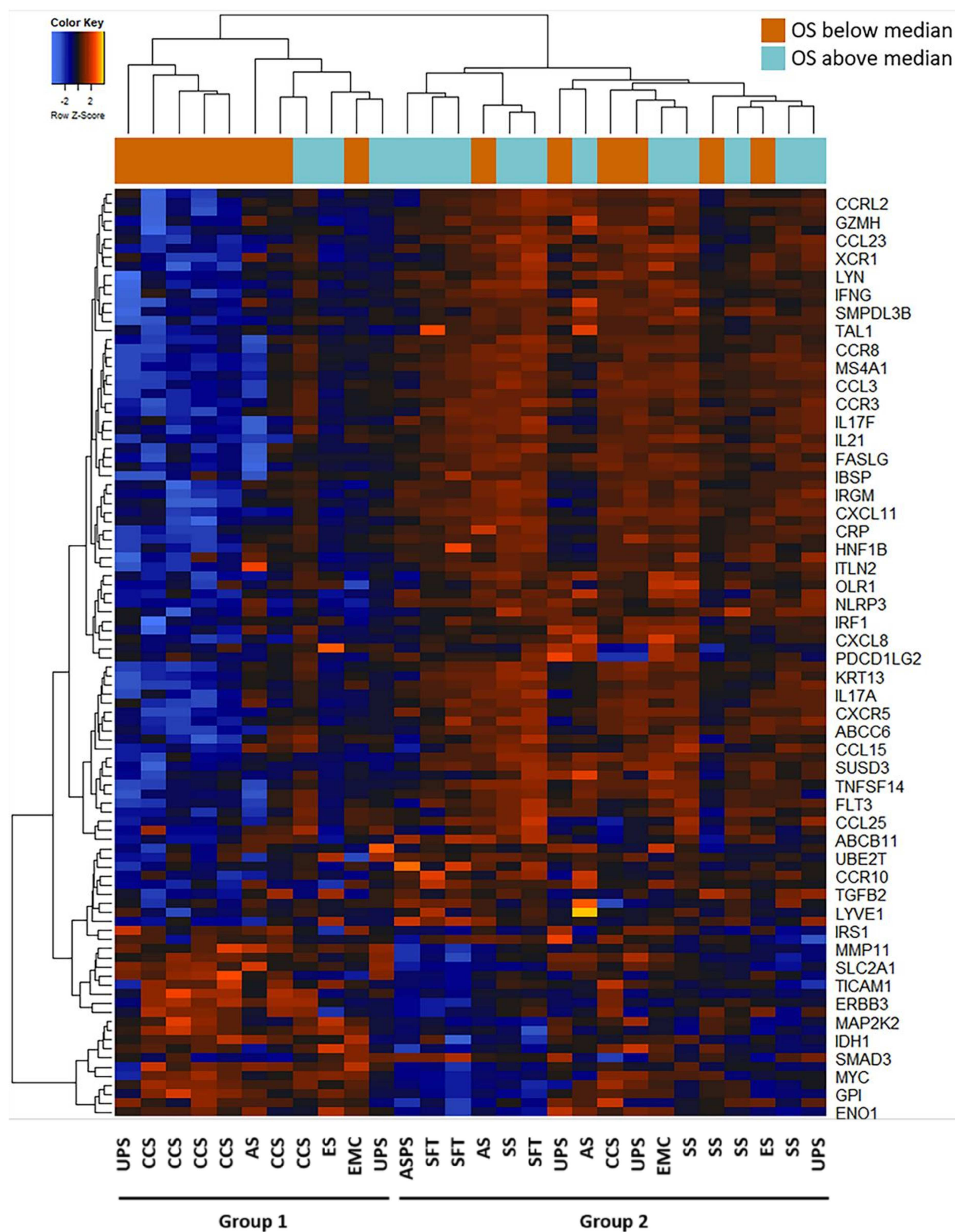

Supplementary Figure 1 – Hierarchical clustering of gene expression profiling, considering only the transcripts related with immune response in both tumor and its surrounding microenvironment, that have a significant impact in overall survival (OS). Two groups with different molecular profile can be identified. AS: angiosarcoma; ASPS: alveolar soft-part sarcoma; CCS: clear cell sarcoma; EMC: extraskeletal myxoid chondrosarcoma; ES: epithelioid sarcoma; SFT: solitary fibrous tumor; SS: synovial sarcoma and UPS: undifferentiated pleomorphic sarcoma.

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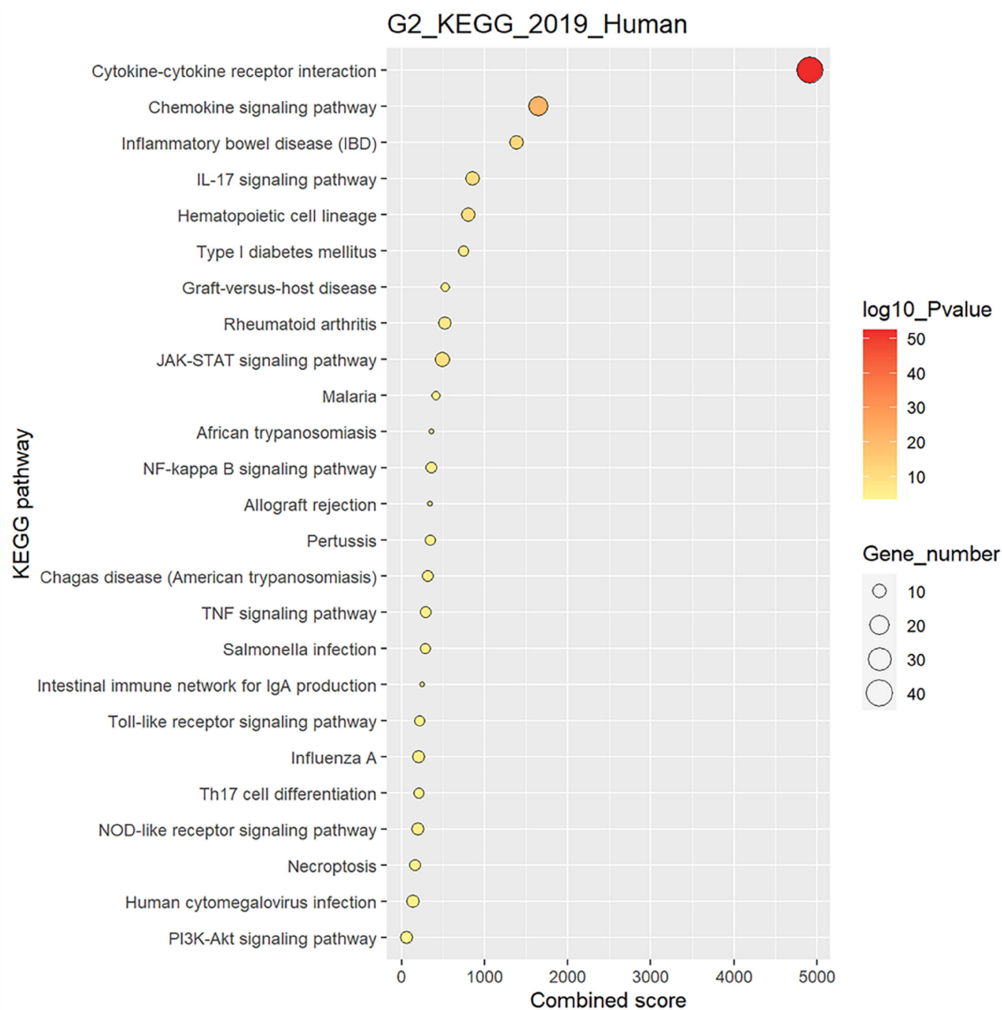
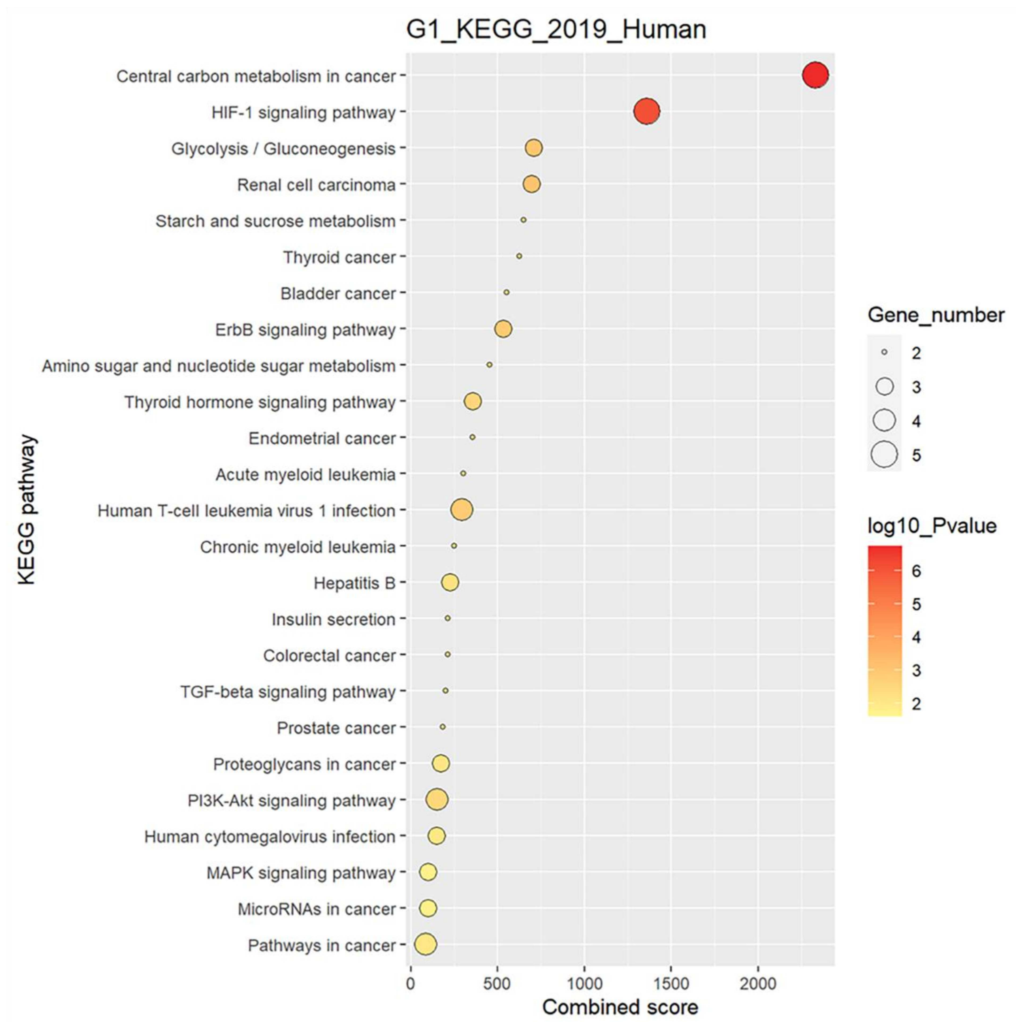


Supplementary Figure 2 – Enrichment analysis in KEGG Human Pathways of selected genes overexpressed in Group 2 (G2). The score obtained in the enrichment analysis performed using enrichR is represented by KEGG pathway. Size of the circle corresponds with the number of genes enriched in the KEGG pathway. The absolute value of the adjusted p-value after log transformation is remarked in a color gradient, being the redder the more significant values.



Supplementary Figure 3 – Enrichment analysis in KEGG Human Pathways of selected genes overexpressed in Group 1 (G1). The score obtained in the enrichment analysis performed using enrichR is represented by KEGG pathway. Size of the circle corresponds with the number of genes enriched in the KEGG pathway. The absolute value of the adjusted p-value after log transformation is remarked in a color gradient, being the redder the more significant values.

Supplementary Table 1 – Toxicity profile of phase II part

PHASE II (N = 52)					
Type of adverse event (n=52)	**Any grade N (%)**	**Grade 1-2 N (%)**	**Grade 3 N (%)**	**Grade 4 N (%)**	**Grade 5 N (%)**
Hematological toxicity					
Leukopenia	24 (46.2%)	22 (42.3%)	2 (3.8%)	0	0
Neutropenia	23 (44.2%)	17 (32.7%)	6 (11.5%)	0	0
Anemia	19 (36.5%)	19 (36.5%)	0	0	0
Thrombocytopenia	16 (30.8%)	13 (25.0%)	2 (3.8%)	1 (1.9%)	0
Lymphopenia	10 (19.2%)	8 (15.4%)	2 (3.8%)	0	0
Febrile neutropenia	1 (1.9%)	0	1 (1.9%)	0	0
Non-hematological toxicity					
Fatigue	33 (63.5%)	28 (53.8%)	5 (9.6%)	0	0
AST increased	25 (48.0%)	19 (36.5%)	6 (11.5%)	0	0
ALT increased	24 (46.2%)	15 (28.8%)	9 (17.3%)	0	0
Hypertension	24 (46.2%)	19 (36.5%)	5 (9.6%)	0	0
Diarrhea	22 (42.3%)	20 (38.5%)	2 (3.8%)	0	0
Mucositis oral	22 (42.3%)	21 (40.4%)	1 (1.9%)	0	0
ALP increased	10 (19.2%)	10 (19.2%)	0	0	0
Nausea	10 (19.2%)	10 (19.2%)	0	0	0
Hypothyroidism	10 (19.2%)	10 (19.2%)	0	0	0
Vomiting	10 (19.2%)	10 (19.2%)	0	0	0
Anorexia	8 (15.4%)	7 (13.5%)	1 (1.9%)	0	0
Dysgeusia	8 (15.4%)	8 (15.4%)	0	0	0
Arthralgia	7 (13.5%)	7 (13.5%)	0	0	0
Edema	7 (13.5%)	7 (13.5%)	0	0	0
Dyspepsia	6 (11.5%)	6 (11.5%)	0	0	0
Myalgia	6 (11.5%)	6 (11.5%)	0	0	0
Skin/subcutaneous tissue disorders	6 (11.5%)	6 (11.5%)	0	0	0
GGT increased	5 (9.6%)	4 (7.7%)	1 (1.9%)	0	0
Palmar-plantar erythrodysesthesia syndrome	5 (9.6%)	5 (9.6%)	0	0	0
Weight loss	5 (9.6%)	5 (9.6%)	0	0	0
Creatinine increased	4 (7.7%)	2 (3.8%)	1 (1.9%)	1 (1.9%)	0
Body Pain	4 (7.7%)	3 (5.8%)	1 (1.9%)	0	0
Epistaxis	4 (7.7%)	4 (7.7%)	0	0	0
Hair color changes	4 (7.7%)	3 (5.8%)	1 (1.9%)	0	0
Bronchopulmonary hemorrhage	3 (5.8%)	2 (3.8%)	1 (1.9%)	0	0
Dry mouth	3 (5.8%)	3 (5.8%)	0	0	0
Heart failure	3 (5.8%)	3 (5.8%)	0	0	0
Hypophosphatemia	3 (5.8%)	2 (3.8%)	1 (1.9%)	0	0

Supplementary Table 2 – Genes with different mean values in both groups clustered taking into account medial overall survival

gene	Statistic	dm	p.value	FDR	Bonferroni	UP/DOWN
SUSD3	-7,08	-1,98	1,63E-07	8,01E-06	1,66E-05	DOWN
CXCL11	-7,05	-2,12	1,72E-07	8,01E-06	1,75E-05	DOWN
CASP5	-6,82	-2,46	3,06E-07	8,01E-06	3,12E-05	DOWN
CXCR5	-6,81	-2,29	3,14E-07	8,01E-06	3,20E-05	DOWN
NLRP3	-6,45	-1,70	7,71E-07	1,57E-05	7,87E-05	DOWN
CD79A	-6,16	-2,09	1,62E-06	2,18E-05	1,65E-04	DOWN
CCL23	-6,10	-1,90	1,92E-06	2,18E-05	1,95E-04	DOWN
MS4A1	-6,09	-2,75	1,94E-06	2,18E-05	1,97E-04	DOWN
IL21	-6,07	-2,64	2,03E-06	2,18E-05	2,07E-04	DOWN
CXCR2	-6,05	-2,13	2,14E-06	2,18E-05	2,18E-04	DOWN
FASLG	-5,98	-2,45	2,61E-06	2,42E-05	2,66E-04	DOWN
IL5	-5,84	-2,85	3,69E-06	3,14E-05	3,77E-04	DOWN
IRGM	-5,67	-1,95	5,85E-06	4,34E-05	5,97E-04	DOWN
CXCR1	-5,64	-2,36	6,28E-06	4,34E-05	6,40E-04	DOWN
CCR8	-5,62	-2,53	6,58E-06	4,34E-05	6,72E-04	DOWN
CCL8	-5,59	-2,56	7,21E-06	4,34E-05	7,36E-04	DOWN
XCL1	-5,58	-2,73	7,24E-06	4,34E-05	7,39E-04	DOWN
CXCR6	-5,55	-1,44	7,86E-06	4,45E-05	8,02E-04	DOWN
TNFSF14	-5,53	-2,70	8,31E-06	4,46E-05	8,48E-04	DOWN
IL1B	-5,44	-2,25	1,06E-05	5,15E-05	1,08E-03	DOWN
IL22RA2	-5,42	-2,73	1,10E-05	5,15E-05	1,12E-03	DOWN
IL17F	-5,40	-2,46	1,19E-05	5,15E-05	1,21E-03	DOWN
CD160	-5,38	-2,78	1,24E-05	5,15E-05	1,27E-03	DOWN
CCR3	-5,37	-2,19	1,26E-05	5,15E-05	1,29E-03	DOWN
CCL7	-5,37	-2,40	1,26E-05	5,15E-05	1,29E-03	DOWN
IL5RA	-5,25	-2,22	1,72E-05	6,75E-05	1,76E-03	DOWN
MMP11	5,21	2,98	1,92E-05	7,24E-05	1,95E-03	UP
LYN	-5,15	-2,00	2,27E-05	8,01E-05	2,32E-03	DOWN
IL1A	-5,15	-2,12	2,28E-05	8,01E-05	2,32E-03	DOWN
SMPDL3B	-5,11	-2,07	2,54E-05	8,24E-05	2,59E-03	DOWN
CCL3	-5,10	-2,63	2,60E-05	8,24E-05	2,65E-03	DOWN
TNFRSF9	-5,09	-2,51	2,66E-05	8,24E-05	2,71E-03	DOWN
IL3	-5,08	-2,52	2,71E-05	8,24E-05	2,76E-03	DOWN
LIPE	-5,08	-2,40	2,75E-05	8,24E-05	2,80E-03	DOWN
AICDA	-5,02	-2,12	3,21E-05	9,37E-05	3,28E-03	DOWN
GZMH	-4,93	-1,94	4,02E-05	1,13E-04	4,10E-03	DOWN
RBX1	4,93	0,70	4,09E-05	1,13E-04	4,17E-03	UP
CSF3	-4,77	-2,73	6,13E-05	1,65E-04	6,26E-03	DOWN
IFNG	-4,75	-2,14	6,44E-05	1,68E-04	6,56E-03	DOWN
LTA	-4,71	-2,00	7,29E-05	1,86E-04	7,43E-03	DOWN

TAL1	-4,68	-2,60	7,78E-05	1,94E-04	7,94E-03	DOWN
IL7	-4,66	-1,85	8,33E-05	2,01E-04	8,50E-03	DOWN
CCR9	-4,65	-2,16	8,48E-05	2,01E-04	8,65E-03	DOWN
CD274	-4,63	-1,71	8,89E-05	2,02E-04	9,07E-03	DOWN
IL17A	-4,63	-2,17	8,92E-05	2,02E-04	9,10E-03	DOWN
CXCL8	-4,58	-2,09	1,01E-04	2,23E-04	1,03E-02	DOWN
WNT7B	-4,56	-1,95	1,06E-04	2,31E-04	1,09E-02	DOWN
CRP	-4,54	-2,93	1,14E-04	2,42E-04	1,16E-02	DOWN
IL19	-4,53	-1,88	1,16E-04	2,42E-04	1,18E-02	DOWN
IFNB1	-4,47	-2,39	1,38E-04	2,81E-04	1,41E-02	DOWN
KRT13	-4,45	-2,14	1,43E-04	2,85E-04	1,45E-02	DOWN
ABCC6	-4,37	-2,39	1,78E-04	3,50E-04	1,82E-02	DOWN
CCRL2	-4,31	-1,81	2,09E-04	4,02E-04	2,13E-02	DOWN
SLC2A1	4,22	2,30	2,62E-04	4,96E-04	2,68E-02	UP
TNF	-4,21	-2,17	2,73E-04	5,06E-04	2,78E-02	DOWN
CCR7	-4,18	-2,04	2,88E-04	5,25E-04	2,94E-02	DOWN
NOD2	-4,18	-1,27	2,95E-04	5,29E-04	3,01E-02	DOWN
S100B	4,07	3,15	3,92E-04	6,89E-04	4,00E-02	UP
CD55	4,03	2,24	4,35E-04	7,52E-04	4,44E-02	UP
OSM	-3,91	-1,82	5,86E-04	9,95E-04	5,97E-02	DOWN
HNF1B	-3,87	-2,66	6,64E-04	1,10E-03	6,77E-02	DOWN
XCR1	-3,86	-1,46	6,66E-04	1,10E-03	6,80E-02	DOWN
PRR15L	-3,77	-2,04	8,41E-04	1,36E-03	8,58E-02	DOWN
IRF1	-3,75	-1,63	9,00E-04	1,43E-03	9,18E-02	DOWN
FLT3	-3,72	-1,90	9,55E-04	1,50E-03	9,75E-02	DOWN
IBSP	-3,71	-2,18	1,00E-03	1,55E-03	1,02E-01	DOWN
GPI	3,59	1,14	1,34E-03	2,04E-03	1,36E-01	UP
LTB	-3,51	-1,74	1,64E-03	2,46E-03	1,67E-01	DOWN
CCR10	-3,47	-1,46	1,82E-03	2,70E-03	1,86E-01	DOWN
MIF	3,42	1,51	2,06E-03	3,00E-03	2,10E-01	UP
LAG3	-3,29	-1,99	2,85E-03	4,10E-03	2,91E-01	DOWN
CXCL3	-3,23	-1,78	3,37E-03	4,78E-03	3,44E-01	DOWN
ERBB3	3,20	3,19	3,63E-03	5,07E-03	3,70E-01	UP
ITLN2	-3,07	-2,09	4,92E-03	6,78E-03	5,01E-01	DOWN
IDH1	3,02	0,74	5,55E-03	7,55E-03	5,66E-01	UP
FOXP3	-2,98	-1,82	6,18E-03	8,30E-03	6,31E-01	DOWN
OLR1	-2,94	-1,21	6,88E-03	9,11E-03	7,02E-01	DOWN
MAP2K2	2,85	0,70	8,47E-03	1,11E-02	8,63E-01	UP
IL16	-2,79	-1,39	9,72E-03	1,26E-02	9,92E-01	DOWN
CCL15	-2,57	-1,42	1,64E-02	2,09E-02	1,00E+00	DOWN
ENO1	2,48	1,25	1,97E-02	2,48E-02	1,00E+00	UP
CREB5	2,32	1,11	2,82E-02	3,50E-02	1,00E+00	UP
HK2	2,26	1,18	3,25E-02	4,00E-02	1,00E+00	UP
MYC	2,22	1,49	3,51E-02	4,27E-02	1,00E+00	UP

Dm: difference of means; Down and up means that the gene is over or infra-expressed in group 2, respectively.