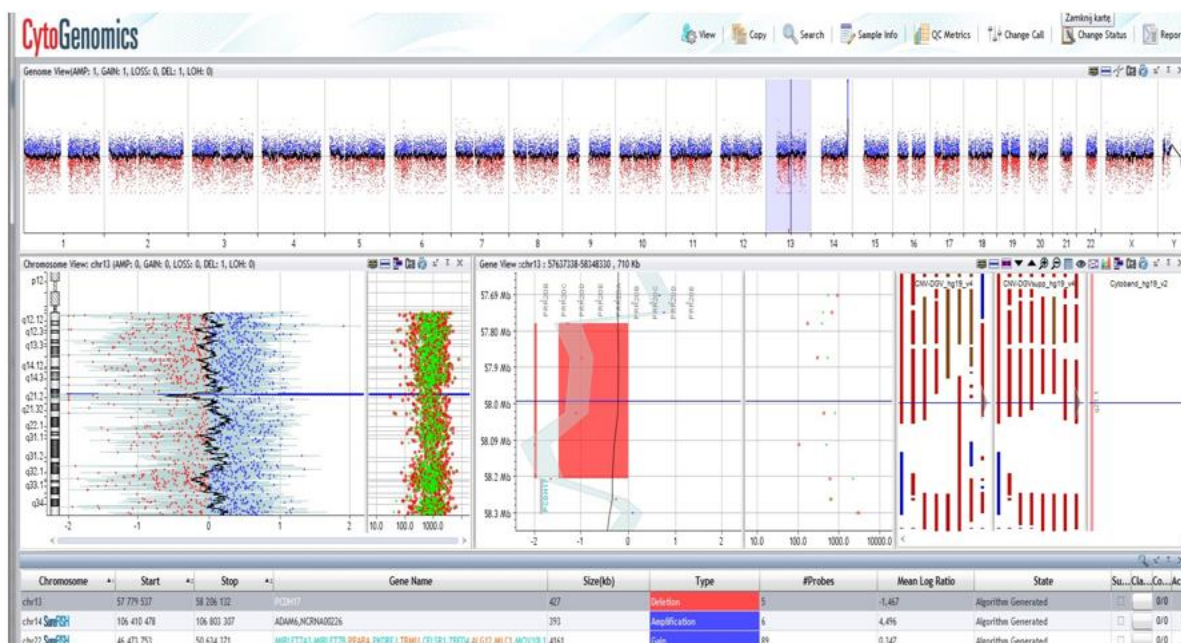


Pathway	Up-regulated genes	Down regulated genes
Inflammation mediated by chemokine and cytokine signaling	<i>RRAS2, ITGB2, PRKACB, ITGAM, PTGS2</i>	<i>PRKCZ, MYLK</i>
Wnt signaling	<i>TCF7, MYCN</i>	<i>CTBP2, PRKCZ, PCDH9, FZD2</i>
Angiogenesis	<i>TCF7</i>	<i>EFNB2, PRKCZ, FZD2</i>
Gonadotropin releasing hormone receptor	<i>TCF7</i>	<i>DUSP1, PRKCZ, ANXA5</i>
Endothelin signaling	<i>PRKACB, PTGS2</i>	<i>PRKCZ</i>
Integrin signalling	<i>ITGB2, ITGAM, ACTN1</i>	-
T cell activation	<i>CD28, CD3D</i>	<i>HLA-DOA</i>

Comperative genomic analysis



Highlights:

- I. Groups of genes are primarily responsible for a genetic lack of sensitivity to CP in AL.
- II. Recurrent chromosomal aberrations are associated with *in vitro* resistance to CP.
- III. Simultaneous decrease in expression for *ABCG1*, *PCDH9*, and overexpression for *DUSP2*, *ITGAM*, *ANXA1*, *FGR*, *SERPINA1*, *HK3* and *RETN* were found in the CP- resistant blasts.

- IV. Overexpression of genes on chromosomes 1,2,10,12 and a reduction in the level of expression for genes located on chromosomes 20,21,22 is correlated with CP-resistance.