**Effects of compounds on respiration and glycolytic rates in treated and untreated tumorigenic Jurkat cells**

The efficiency of the inhibitors was also tested by measuring respiratory flow, maximal respiratory rate, glycolytic rate, and glycolytic capacity in Jurkat cells. Bioenergetic studies were performed using the Seahorse XFp assay (Agilent Technology), which allows non-destructive measurements of the oxygen consumption rate (OCR) and extracellular acidification rate (ECAR) of the cells tested.

First, basal glycolytic respirations and basal glycolytic fluxes were determined by measuring the OCR and ECAR values respectively, in the untreated Jurkat cells (vehicle) and the cells treated with cmpd No. 9 or No. 30.

Lower respiration levels were detected in the cells treated by cmpds No. 9 and 30 compared to the untreated cells. Similarly, measurements of basal glycolytic rates in the media with added cmpds were observed however, stronger statistical significances were obtained from extracellular acidification rates measurements compared to respiration.

After 20 minutes of incubation, immediately after the samples for respiration and glycolysis have been taken, Oligomycin A, an inhibitor of mitochondrial ATP synthase, and FCCP, a protonophore causing a breakdown of the mitochondrial inner membrane that disrupts hydrogen ions transport were added to the medium. Dysfunctional mitochondrial oxidative phosphorylation is supposed to induce glycolytic stress which enables the determination of maximal respiration and glycolytic capacity.

Samples collected 6.5 minutes after the first sampling, showed a stronger increase of respiration and acidification in the cells treated with cmpd No. 9 or No. 30 compared to the vehicle. However, levels of maximal oxygen consumption and glycolytic capacities in the treated cells remained lower than those of the untreated cells (**Fig. 7**). The graphics of the analysis in time of the experiments are shown in the Supplementary materials (S13 Fig.).

It is worth noting that tumorigenic Jurkat calls treated by cmpds can reduce oxigen consumption rate and (ECAR) exocellular acidification rate and inhibit cancer-specific PFK1 activities that consequently subsidization of deleterious uncontrolled energy metabolize in treated tumorigenic cells.