oncogenic pathways was also assessed. The performance of previously identified immune signatures as the Immunologic Constant of Rejection (2,3), which captures an active Th1/cytotoxic response associated with favorable prognosis and responsiveness to immunotherapy, was also checked within each tumor subtype.

**Results** We found 5 main modules, in agreement with results obtained in adult solid tumors: Wound Healing, TGF-B signaling, IFN-G signaling, Macrophages and Lymphocytes (figure 1). These 5 modules clustered pediatric patients into 6 immune subtypes: S1-S6 with distinct survival (S2 vs S4, p=0.0044, adjusted for cancer type). S2 cluster has the best overall survival and characterized by low enrichment of wound healing signature, high Th1, low Th2 and high expression of HLA1 and HLA2, while the opposite holds true for cluster S4 with the worst survival and highest enrichment of wound healing signature, high Th2, and low Th1. The S6 cluster is characterized by highest enrichment of lymphocyte signature, the highest expression of immune checkpoints accompanied by elevated expression of exhaustion markers, and an unpolarized immune response with high abundance of macrophages. Additionally, pan-cancer, the upregulation of WNT-Beta-catenin pathway is associated with adverse outcome and lack of T-cell infiltration. In the peritumor cancer analysis, ICR is associated with better survival in osteosarcoma and with worse survival in Wilms' tumors, similarly with what observed in adult kidney's cancer despite the different embryological origin.

**Conclusions** We demonstrated that pediatric solid cancers can be classified according to their immune disposition, unveiling unexpected similarity with adults' tumors. Immunological parameters might be explored to refine diagnostic and prognostic biomarkers and to identify potential immune responsive analyses. This is the first pan-cancer immunogenomic analysis in children.

**REFERENCES**


**Consent** Written informed consent was obtained from the patient to use the results of this study for educational purposes including publications. A copy of the written consent is on file and available for review by the Editor of this journal.

**METABOLIC REQUISITES FOR T CELL PROTEIN TRANSLATION IN TUMORS**

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**Background** T cells are a secretory immune subset with the capacity to control solid tumors. Protein translation is of paramount importance in CD8 T cells, controlling proliferation, stimulation and lineage fate.

**Methods** Here, we used both the fluorescent analogy of methionine homopropargylglycine (HPG) incorporation assay and O-propargyl-puromycin (OPP) method which enters the A-site of the ribosome and effectively labels and terminates nascent polypeptide chains to monitor protein synthesis in mouse and human tumors. Moreover, we employed label free quantitative proteomics (LFQ), lipidomics, metabolic analysis, and in vivo animal modeling to elucidate mechanisms of protein translation in antitumor immunity.

**Results** We found that canonical protein synthesis is restricted in endogenous CD8 tumors infiltrating lymphocytes (TILs) by the tumor microenvironment (TME). Proteomic analysis revealed that glucoseogenesis and B-oxidation of fatty acids (FAO) were upregulated in CD8 T cells under tumor stress but these metabolic sources were unable to support translation in the TME. Further, we discovered that glucose metabolism and mammalian target of rapamycin complex 1 (mTORC1) preferentially hinder protein synthesis in CD8 TILs. These data enabled the discovery that proteasomal protein...