cellular components of the immune system. While immunotherapies have recently revolutionized cancer therapy, they have shown so far little therapeutic success in glioblastoma patients. To enhance the efficacy of new strategies, we need to better understand the immunogenic status of glioblastoma cells and their cross-talk with immune cells in different microenvironmental niches. MATERIAL AND METHODS: We assessed expression of molecules related to antigen processing and presentation and costimulatory checkpoints in patient-derived glioblastoma organoids and in a series of glioblastoma patient-derived organoids, 3D stem-like cultures and adherent cell lines under varying microenvironmental conditions (varying oxygen levels, inflammation). We further established an allogeneic co-culture protocol for glioblastoma organoids with immune cells isolated from HLA-matched donor blood, allowing for the functional assessment of the crosstalk between tumor and immune cells. RESULTS: Analysis of a large cohort of patient tumors and patient-derived glioblastoma preclinical models shows inter-patient heterogeneity at the level of components of microtumor-infiltrating lymphocyte complex (MHC-I, MHC-II, B7, CD40, TGF-β, IFN-γ). Glioblastoma cells in general express MHC-I machinery, albeit at different levels. MHC-II and immune checkpoints are variably expressed across glioblastoma cells. Different tumor microenvironment conditions, including hypoxia and interferon-γ, impact the expression of immune-related molecules. Upon co-culture, HLA-matched donor-derived T cells integrate well into the core of glioblastoma tumor organoids and display reciprocal cross-talk with tumor cells. CONCLUSION: Assessing antigen presentation and immune cell responses at the functional level are key to improving specific responses to immunotherapies. Advanced glioblastoma organoids incorporating the immune compartment appear as clinically-relevant models for ex vivo efficacy studies.

P12.15.B. ASTROCYTE IMMUNOMETABOLIC REGULATION OF THE GLIOBLASTOMA MICROENVIRONMENT DRIVES TUMOR PATHOGENICITY

R. Perelrozen, B. Philippou, N. Budick-Harmelin, T. Chernobylbfsky, K. Rotem, A. Ron, D. Shimon, A. Teslser, O. Adir, A. Gaoi-Yogev, T. Michael, A. Mager, E. Ruppin, L. Mann, Tel Aviv University, Tel Aviv, Israel; National Cancer Institute, Bethesda, MD, United States.

BACKGROUND: Malignant brain tumors are the cause of a disproportionate level of morbidity and mortality among cancer patients, an unfortunate statistic that has remained constant for decades. Despite considerable advances in the molecular characterization of these tumors, targeting the cancer cells has yet to produce significant advances in treatment. An alternative strategy is to target cells in the glioblastoma microenvironment, such as tumor associated astrocytes. Astrocytes control multiple processes in health and disease, ranging from maintaining the brain's metabolic homostasis, to modulating neuroinflammation. However, their role in glioblastoma pathogenicity is not well understood. MATERIAL AND METHODS: Immuno-competent mice were implanted with murine glioma cell lines and the role of astrocytes in the tumor pathogenicity was analyzed, and further investigating using in-vitro co-cultures. RESULTS: Here we report that depletion of reactive astrocytes regresses glioblastoma and prolongs mouse survival. Analysis of the tumor-associated astrocyte transcriptome, revealed that astrocytes initiate transcriptional programs that shape the immune and metabolic compartment in the glioma microenvironment. Specifically, their expression of CCL2 and CSF1 governs the recruitment of tumor-associated macrophages and promotes a pro-tumorigenic macrophage phenotype. Concomitantly, we demonstrate that astrocyte-derived cholesterol is key to glioma cell survival, and that targeting astrocyte cholesterol efflux, via ABCA1, halts tumor progression. In summary, astrocytes control glioblastoma pathogenicity by re-programming the immunological properties of the tumor microenvironment and supporting the non-oncogenic metabolic dependency of glioblastoma on cholesterol. CONCLUSION: These findings suggest that targeting astrocyte immunometabolic signaling may help treat this uniformly lethal brain tumor.

P13.02.B. FULLY AUTOMATED SEGMENTATION AND VOLUMETRIC MEASUREMENT OF INTRACRANIAL MENINGIOMA USING DEEP LEARNING

H. Kang, P. Kim, N. Wotan, K. Lee, Y. Koo, S. Choi, Y. Kim, K. Kim, S. Park, C. Park; Seoul National University Hospital, Jongno-gu, Korea, Republic of; MEDICALIP Co. Ltd, Jongno-gu, Korea, Republic of.

BACKGROUND: Most intracranial meningiomas are small, asymptomatic, and incidentally found tumors. Since the growth of meningioma