expression level of TSPAN12 was negatively correlated with the prognosis in MG patients. Collectively, more studies are needed, but we suggest that TSPAN12 can be used as an alternative biomarker to predict the prognosis of MG patient and help develop MG treatment strategies.

BIOM-39. P-ERK ASSOCIATION WITH OVERALL SURVIVAL IN RECURRENT GBM PATIENTS TREATED WITH INTRACEREBRAL ADMINISTRATION OF PD-1 AND CTLA-4 BLOCKING ANTIBODIES

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INTRODUCTION: Anti-PD-1 immunotherapy induces clinical responses in a subset of glioblastoma (GBM) patients. We previously reported that ERK1/2 phosphorylation (p-ERK) in pre-treatment tumor samples is predictive of overall survival (OS) following adjuvant anti-PD-1 therapy in two independent cohorts of recurrent GBM patients. METHODS: Following the Remark criteria for biomarker validation, we investigated p-ERK as a predictive of OS in 24 evaluable tumor samples of recurrent GBM patients from a clinical trial. These patients underwent intracerebral administration of immune checkpoint inhibitors as part of a phase I clinical trial where intracerebral administration of ipilimumab (10 mg) or ipilimumab (5 mg) and nivolumab (10 mg) followed by postsurgical intravenous nivolumab (10 mg) was evaluated (NCT03233152; Duerinck J, et al. JITC, 2021). We quantified cell density of p-ERK$^+$ cells in tumor regions. For exploratory purposes, patients were divided in 3 groups (n=8 per group) bases on p-ERK cell density. RESULTS: We observed an incremental OS with high p-ERK GBM patients exhibiting a median OS of 81.6 weeks (95% CI 33.86-NA), intermediate p-ERK median OS of 43.1 weeks (95% CI 33.14-NA), and low p-ERK group with a median OS of 19.3 weeks (95% CI 16.14-NA). A Cox proportional hazards model adjusted for age and *IDH* mutant status showed a trend for p-ERK association with favorable OS (HR= 0.77, 95% CI 0.6-=1.01, P=0.056). CONCLUSIONS: While the number of patients analyzed is relatively small, this study suggests the potential predictive power of p-ERK in an independent prospective GBM cohort treated with an alternative and unique administration approach of immune checkpoint blockade.

BIOM-40. COMPARATIVE STUDY OF INFLAMMATORY MARKERS IN THE CEREBROSPINAL FLUID OF PATIENTS WITH GLIOMA AND CENTRAL NERVOUS SYSTEM MALIGNANT LYMPHOMA

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INTRODUCTION: Cytokines in the cerebrospinal fluid (CSF) are useful as markers for primary central nervous system malignant lymphoma (PCBSL). However, there are few reports of CSF markers in glioma. We analyzed and examined glioma and PCNSL CSF inflammation-related mediators. MATERIALS AND METHODS: The patients with glioma and PCNSL who were operated on at Kobe University between 2006 and 2017 and whose histology was diagnosed, and whose CSF was stored at -80 °C before treatment. Using Bio-Plex Pro Human Inflammation Assays (Bio-Rad Laboratories, Inc.), 37 types of inflammation-related molecules such as TNF superfamilies, interferonss, and Interleukines were measured and compared with glioma and PCNSL. RESULTS: The subjects were 53 glioma patients and 24 PCNSL patients. There were 22 types of significant differences between glioma and PCNSL, 18 with high PCNSL and 4 with high glioma. In addition to IL-10, which has been reported so far, the significant higher concentrations in PCNSL was found in Osteopontin, TNFSF8, TNFSF12, TNFSF13, TNFSF13B, TNFSF14, sTNF-R1, sTNF-R2, IL-6Ra, IL-8, IL-12(p40), IL-20, IL-27, sCD163, MMP2, MMP3, CHI3L1. On the other hand, IL-2, Il-12(p70), IL-22, and MMP-1 levels were significant higher in CSF in glioma. Although CD163 is an important molecule as a marker for M2 macrophages, immunostaining of tissues showed no significant difference in infiltration between Glioblastoma and PCNSL. This suggests that M2 macrophages infiltrating PCNSL and Glioblastoma have different activation states. CONCLUSION: PCNSL had higher levels of inflammatory mediators such as TNF-related molecules. PCNSL was significantly higher in sCD163 than in glioblastoma, suggesting different activation of intratumoral M2 macrophages.

BIOM-41. IDENTIFICATION OF GLIOMA CELLS BY PROFILING SINGLE-CELL BIOPHYSICAL PROPERTIES THROUGH MULTICONSTRICTION MICROFLUIDIC CHANNELS

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This study introduces a multicontraction microfluidic channel that can differentiate glioma cells from normal glio cells. As cells pass through successive constriction channels, the incremental velocity and varying size profiles of the cells will be collected, reflecting their biophysical properties. The data of high-dimensional variables were analyzed , including the cell sizes, velocities, and velocity increments. The prediction value is used to represent the difference between two groups using the established classification model from high-dimensional variables. At the same time, we prepared three groups of primary tumor cells from patients with different grades of glioma to verify the efficacy of this classification method. The results show that in this microfluidic channel, the diagnostic model made of cell biophysical properties can well identify glioma cells, which gives a novel method for efficient identification of circulating tumor cells and rapid pathological diagnosis.

BIOM-42. A DEEP LEARNING MODEL FOR AUTOMATED DETECTION AND COUNTING OF TUNNELING NANOTUBES AND CANCER CELLS IN MICROSCOPY IMAGES

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BACKGROUND: Tunneling nanotubes (TNTs) are cellular structures connecting cell membranes and mediating intercellular communication. TNTs are manually identified and counted by a trained investigator; however, this process is time-intensive. We therefore sought to develop an automated approach for quantitative analysis of TNTs. METHODS: We used the convolutional neural network (U-Net) deep learning model to segment phase contrast microscopy images of both cancer and non-cancer cells. Our method was composed of preprocessing and model development. We developed a new preprocessing method to label TNTs on a pixel-wise basis. Two sequential models were employed to detect TNTs. First, we identified the regions of images with TNTs by implementing a classification algorithm. Second, we fed parts of the image classified as TNT-containing into a modified U-Net model to estimate TNTs on a pixel-wise basis. RESULTS: The U-Net model detected 73.3% of human expert-identified TNTs, counted TNTs and cells, and calculated the TNT-to-cell ratio (TCR). We obtained a precision of 0.88, recall of 0.67, and f-1 score of 0.76 on a test data set. The predicted and true TCRs were not significantly different between the training and test data sets. CONCLUSIONS: In summary, we report application of an automated model generated by deep learning and trained to accurately label and detect TNTs and cells imaged in culture. Continued application and refinement of this process will provide a new approach to the analysis of TNTs, which form to connect cancer and other cells. This approach has the potential to enhance the drug screens intended to assess therapeutic efficacy of experimental agents, and to reproducibly assess TNTs as a potential biomarker of response to therapy in cancer.

BIOM-43. THE GENOMIC, TRANSCRIPTOMIC, AND EPIGENOMIC LANDSCAPE OF ISOCITRATE DEHYDROGENASE WILD TYPE GLIOBLASTOMA ACROSS THE AGE CONTINUUM

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