The Canadian Brain Tumour Registry (CBTR) project was established in 2016 with the aim of enhancing infrastructure for surveillance and clinical research to improve health outcomes for brain tumour patients in Canada. We present a national surveillance report on malignant primary brain and central nervous system (CNS) tumours diagnosed in the Canadian population from 2009-2013. Patients were identified through the Canadian Cancer Registry (CCR); an administrative dataset that includes cancer incidence data from all provinces/territories in Canada. Cancer diagnoses are coded using the ICD-O3 system. Tumour types were classified by site and histology using The Central Brain Tumour Registry of the United States definitions. Incidence rates (IR) and 95% confidence intervals (CI) were calculated per 100,000 person-years and standardized to the 2011 census population age-distribution. Overall, 12,115 malignant brain and CNS tumours were diagnosed in the Canadian population from 2009-2013 (IR:8.43;95%CI:8.28,8.58). Of these, 6,845 were diagnosed in males (IR:9.72;95%CI:9.49,9.95) and 5,270 in females (IR:7.20;95%CI:7.00,7.39). The most common histology overall was glioblastoma (IR:4.06;95%CI:3.95,4.16). Among those aged 0-19 years, 1,130 malignant brain and CNS tumours were diagnosed from 2009-2013 (IR:3.36;95%CI:3.16,3.56). Of these, 625 were diagnosed in males (IR:3.32;95%CI:3.34,3.92) and 505 in females (IR:3.08;95%CI:2.81,3.36). The most common histology among the paediatric population was pilocytic astrocytoma (IR:0.73;95%CI:0.64,0.83). The presentation will include: IRs for other histologies, the geographic distribution of cases and a comparison between Canada and the United States.

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Unique Immune Microenvironment in NF2-Fusion Positive Radiation Induced Meningiomas

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Introduction: Radiation-induced meningiomas (RIMs) are increasing in prevalence as cancer patients live longer. Our laboratory has demonstrated that RIMs have a unique genomic landscape compared to sporadic meningiomas. Notably, a subset of RIMs harbor genomic rearrangement resulting in NF2 gene fusion with a nonrecurrent reciprocal gene. We aimed to compare the gene expression of NF2-Fusion and NF2-Wild Type (NF2-WT) RIMs. Methods: RNA sequencing using Illumina HiSeq was performed on 7 NF2-Fusion and 12 NF2-WT RIMs. Short read sequences obtained from sequencing were mapped to reference human genome(hg19). We performed differential expression analysis using edgeR statistical packages. Pathway analysis was performed using Gene Set Enrichment Analysis (GSEA). Immunohistochemistry was performed to validate findings. Results: Principal component analysis revealed that 5/7 of NF2-Fusion RIMs had similar gene expression profiles. One outlier had no chromosome 1p loss like the other NF2-Fusion RIMs. Pathway analysis demonstrated there was an upregulation of immune pathways in NF2-Fusion RIMs. Immunohistochemistry of PD-L1 revealed that 0/7 and 7/7 of NF2-Fusion tumors had positive expression in tumoral and inflammatory cells, respectively. In comparison, 6/12 of RIMs had tumoral and inflammatory cell expression of PD-L1. In addition, there was a higher CD3 lymphocyte infiltration in NF2-WT (42.2 vs. 12.4 number of cells per HPF). Discussion: Preliminary data in our lab demonstrates that NF2-Fusion tumors have a distinct immune microenvironment compared to NF2-WT tumors. Although pathway analysis indicates that NF2-Fusion RIMs have overexpression of immune pathways, immunohistochemistry reveals that inflammatory cells have positive PD-L1 expression, suggestive of immune burnout.