Canada is unknown. We compared cohorts between 2 regional Canadian Cancer Centres for differences in patient factors, treatments, and outcomes. Methods Cohorts were constructed by a hybrid of retrospective chart review and prospective data collection consisting of all consecutive cases eligible for standard treatment. Demographics, pathology, treatment, and outcome data were obtained. Results The two cohorts (Winnipeg n=80 and Calgary n=103) were similar in terms of median age (57 and 56), percent male (62.5% and 63.1%), percent with good performance status (93.8% vs 85.4%) and extent of resection (gross total/sub-total/biopsy: 17.5%/66.3%/16.3% in Winnipeg and 7.8%/68.9%/23.3% in Calgary). Of patients with known MGMT promoter methylation status 28% were methylated in Winnipeg and 58% were methylated in Calgary. Greater than 6 cycles of chemotherapy was given to 42.5% of patients in Winnipeg and 28.1% in Calgary. The most common second line therapies differed: carboplatin and tamoxifen (31.3%) in Winnipeg; low dose temozolomide (26.2%) in Calgary. Significant poor prognostic factors for survival in the combined cohort included age (HR 1.02), extent of resection (sub-total HR 1.7; biopsy HR 8.9) and location (Calgary HR 1.17). Conclusion Comparison of cohorts from different parts of Canada can provide interesting descriptions of patterns of practice. These patterns may be useful in determining opportunities for quality improvement and clinical trial development.

C10 – Session5 1345-1400
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Glioblastoma multiforme in elderly and non-elderly patients in Newfoundland and Labrador: A province-wide six-year analysis.

Current treatments for Glioblastoma (GB), the most aggressive form of primary brain cancer, include surgery, radiation and chemotherapy. TMZ is the most commonly used alkylating agent for GB treatment, but chemoresistance to TMZ is frequently an unsatisfactory treatment outcome. Relaxin Family Peptide Receptor 1 (RXFP1) mediates RLN2-induced cell migration and tissue invasion in many cancer entities including brain cancer. We have discovered RXFP1 expression in GB cells and tissues, but not in normal astrocytes. Down-regulation of RXFP1 in primary GB cells suppressed cell survival, cell invasiveness and induced cell death via a caspase3/7 mediated apoptosis pathway. Importantly, RXFP1 activation enhanced cell survival in primary GB cells treated with TMZ. To elucidate the mechanisms of RXFP1-mediated chemoresistance in GB cells, we identified the RXFP1-mediated up-regulation of anti-apoptotic proteins. In addition, several DNA repair proteins and Base Excision Repair (BER) members were regulated upon RXFP1 activation. Our results suggest that RXFP1 promotes TMZ chemoresistance by enhancing BER function and by suppressing apoptosis, thus, protecting primary GB cells from TMZ-induced DNA damage.

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RXFP1 promotes temozolomide (TMZ) chemoresistance in brain cancer.
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Chloroquine inhibits the malignant phenotype of glioblastoma in vitro
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