# Association of GLTSCR1, ERCC4, NBN, and XRCC1 Polymorphisms with Glioma and Meningioma Risk in a tertiary care hospital.

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**Association of *GLTSCR1*, *ERCC4*, *NBN*, and *XRCC1* Polymorphisms with Glioma and Meningioma Risk in a tertiary care hospital.**

Brain tumors represent a formidable challenge in neuro-oncology, characterized by their diverse cellular origins, aggressive clinical courses, and often dismal prognoses. Among the spectrum of primary brain tumors, **gliomas** and **meningiomas** are primary brain tumours that account for a significant proportion of cancer-related morbidity and mortality(1).. Gliomas, arising from glial cells (astrocytes, oligodendrocytes, and ependymal cells), are notoriously infiltrative and aggressive, with glioblastoma multiforme (GBM) representing the most malignant and common form in adults, accounting for a significant proportion of primary malignant brain tumors (2). Meningiomas, conversely, originate from the arachnoid cap cells of the meninges, the protective membranes surrounding the brain and spinal cord. While predominantly benign (World Health Organization Grade I), their location, size, and potential for recurrence or malignant transformation necessitate careful monitoring and treatment (3). The global incidence of both tumor types, though varying geographically, underscores their substantial public health burden. In India, like other parts of the world, brain tumors contribute significantly to neurological morbidity and mortality, placing a unique strain on healthcare resources (4).

The etiology of both meningiomas and gliomas is complex and multifactorial, involving a convoluted interplay of genetic predisposition and environmental exposures. While ionizing radiation is a well-established risk factor for both, particularly meningiomas, the vast majority of cases occur sporadically, hinting at a strong underlying genetic component (5). This notion is further supported by the identification of various germline mutations and single nucleotide polymorphisms (SNPs) associated with increased susceptibility. A critical area of investigation into this genetic predisposition lies in the realm of **DNA repair mechanisms**. The integrity of the human genome is under constant assault from endogenous metabolic byproducts, reactive oxygen species, and exogenous agents like UV radiation and chemical carcinogens. To counteract this relentless damage, cells are equipped with an intricate network of DNA repair pathways, including

nucleotide excision repair (NER), base excision repair (BER), mismatch repair (MMR), and double-strand break repair (DSBR) via homologous recombination (HR) or non-homologous end joining (NHEJ) (6). These pathways collectively ensure genomic stability, prevent the accumulation of mutations, and thus safeguard against malignant transformation.

Polymorphisms within genes encoding DNA repair proteins can subtly alter the efficiency or fidelity of these crucial repair processes. While many polymorphisms are benign, those occurring in functionally important regions of DNA repair genes can lead to a reduced capacity to mend DNA damage, rendering individuals more susceptible to various diseases, including cancer (7). For instance, variations in genes like *XRCC1* (involved in BER), *XPC* (involved in NER), *MGMT* (an enzyme that directly repairs O6-methylguanine adducts), and various genes involved in DSBR pathways are frequently studied in the context of cancer susceptibility. The concept is that individuals carrying certain less efficient polymorphic variants might accumulate DNA damage at a higher rate, consequently increasing their risk of developing cancer over time.

Investigating the association of these DNA repair polymorphisms with brain tumors holds significant **clinical utility**, particularly in identifying individuals at higher genetic risk, potentially leading to improved screening, surveillance, and personalized prevention strategies..Differences in allele frequencies of specific DNA repair polymorphisms, as well as their interactions with local environmental carcinogens, could influence brain tumor incidence and prognosis within the Indian subcontinent (8).Understanding the associations could pave the way for risk stratification models tailored to the Indian demographic, enabling clinicians to identify high-risk individuals who might benefit from early detection protocols or targeted chemopreventive measures. Furthermore, these genetic insights refine tumor biology, guiding novel therapies and predicting treatment response. As studied, polymorphisms affecting *MGMT* expression are known to influence response to temozolomide in glioma patients, highlighting the direct clinical relevance of these genetic variations (1). Therefore, a comprehensive exploration of DNA repair polymorphisms in meningioma and glioma within the Indian population is not merely an academic exercise but a critical step towards enhancing precision medicine in neuro-oncology.

The present study aims to pinpoint the specific DNA repair gene polymorphisms and their frequencies linked to meningioma in our patient group. Also, identifies associations across all

meningioma subtypes and evaluate if these single nucleotide polymorphisms (SNPs) can serve as future diagnostic tools.

**Methodology**

This **hospital-based case-control study** was conducted from **January 2023 to June 2024** at the neurosurgery department of a tertiary care hospital in South India. We included **53 glioma cases, 46 meningioma cases, and 98 healthy controls**, all adults over 18 years, diagnosed clinically and radiologically. Patients with other tumors or those unwilling to participate were excluded. The study protocol received approval from the Institutional Ethics Committee, Andhra Medical College, and informed consent was obtained from all participants.

Diagnoses were confirmed via **MRI and biopsy**, with **biochemical investigations.** Patients presenting with meningioma features, meeting inclusion criteria, will provide written informed consent. Following detailed clinical assessments, **3ml EDTA blood** will be collected and stored at -20°C for DNA analysis.

**DNA isolation and PCR amplification** for specific DNA repair gene polymorphisms performed via **RFLP using a thermal cycler in the Multidisciplinary Research Unit of Andhra Medical College**. We will analyze **ERCC4 rs1800067, GLTSCR1 rs1035938, NBN rs1805794, and XRCC1 rs25487**.

**Results**

This section presents the key findings of the study, detailing the demographic characteristics of the participant groups, the specific single nucleotide polymorphisms (SNPs) investigated, and their associations with glioma and meningioma risk.

**SNPs in DNA Repair Genes**

Table 1 provides an overview of the DNA repair genes and the specific SNPs investigated in this study, including their types, base changes, and chromosomal locations[1].

**Table 1: SNPs in DNA repair genes** [2]

| Type of DNA Repair | Gene Symbol | Gene Name | SNP ID | Base Change | Chromosome Location |
|---|---|---|---|---|---|
| Base excision repair | XRCC1 | X ray repair cross complementing gene | Rs25487 | A/G | 19q13.2 |
| Double strand break repair | NBN/NBS1 | Nibrin | Rs1805794 | G/C | 8q21 |
| Nucleotide excision repair | ERCC4 | Excision repair cross complementing group | Rs1800067 | G/A | 16p13.5 |
| | GLTSCR1 | Glioma tumour suppressor | Rs1035938 | C/T | 19q13.3 |

**Demographic Details of Study Participants**

The demographic characteristics of glioma and meningioma cases and their respective controls are presented in Tables 2 and 3. Both tumor groups showed a balanced gender distribution[3].

**Table 2: Demographic Details for Glioma**

| Characteristic | Glioma Cases | Controls |
|---|---|---|
| Male | 27 | 56 |
| Female | 23 | 42 |
| 18-39 years | 13 | 15 |
| 40-59 years | 20 | 63 |
| 60-90 years | 17 | 20 |

**Table 3: Demographic Details for Meningioma**

| Characteristic | Meningioma Cases | Controls |
|---|---|---|
| Male | 26 | 56 |
| Female | 24 | 42 |
| 18-39 years | 1 | 15 |
| 40-59 years | 26 | 63 |
| 60-90 years | 23 | 20 |

### Association between Polymorphisms and Risk of Glioma

Table 4 summarizes the associations between the investigated SNPs and the risk of glioma, showing the genotype distributions, odds ratios (OR), and p-values for various inheritance models[6].

**Table 4: Association between Polymorphisms and Risk of Glioma**

**Association between Polymorphisms and Risk of Meningioma**

| Gene | SNP ID | Model | Genotype | Cases | Controls | OR | P-value |
|---|---|---|---|---|---|---|---|
| GLTSCR1 | rs1035938 | Dominant | AA + AG vs GG | 33 | 86 | 2.47 | 0.012 |
| | | Co-dominant | AA vs AG vs GG | 33 | 86 | 2.32 | 0.028 |
| | | Recessive | AA vs AG + GG | 33 | 86 | 1.47 | 0.464 |
| | | Over-dominant | AG vs AA + GG | 10 | 109 | 2.59 | 0.031 |
| | | Log-additive | - | - | - | 2.47 | 0.028 |
| ERCC4 | rs1800067 | Dominant | TT + TC vs CC | 31 | 87 | 2.35 | 0.020 |
| | | Co-dominant | TT vs TC vs CC | 31 | 87 | 2.18 | 0.032 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | Recessive | TT vs TC + CC | 31 | 87 | 2.64 | 0.024 |
| | | Over-dominant | TC vs TT + CC | 16 | 102 | 1.20 | 0.794 |
| | | Log-additive | - | - | - | 2.35 | 0.032 |
| NBN | rs1805794 | Dominant | GG vs GA + AA | 27 | 6 | 7.24 | <0.001 |
| | | Co-dominant | GG vs GA vs AA | 27 | 6 | 5.40 | <0.001 |
| | | Recessive | GG + GA vs AA | 22 | 24 | 2.18 | 0.030 |
| | | Over-dominant | GG + AA vs GA | 48 | 5 | 2.16 | 0.144 |
| | | Log-additive | - | - | - | 4.05 | <0.001 |
| XRCC1 | rs25487 | Dominant | CC vs CT + TT | 23 | 18 | 6.74 | <0.001 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | Co-dominant | CC vs CT vs TT | 23 | 18 | 5.30 | <0.001 |
| | | Recessive | CC + CT vs TT | 33 | 21 | 6.05 | <0.001 |
| | | Over-dominant | CC + TT vs CT | 43 | 10 | 0.54 | 0.190 |
| | | Log-additive | - | - | - | 5.20 | <0.001 |

Table 5 presents the associations between the investigated SNPs and the risk of meningioma, including genotype distributions, odds ratios (OR), and p-values for various inheritance models[8].

**Table 5: Association between Polymorphisms and Risk of Meningioma**

| **Gene** | **SNP ID** | **Model** | **Genotype** | **Cases** | **Controls** | **OR** | **P-value** |
|---|---|---|---|---|---|---|---|
| GLTSCR1 | rs1035938 | Dominant | AA + AG vs GG | 33 | 86 | 7.62 | <0.001 |
| | | Co-dominant | AA vs AG vs GG | 33 | 86 | 4.88 | <0.001 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | Recessive | AA vs AG + GG | 33 | 86 | 9.81 | <0.001 |
| | | Over-dominant | AG vs AA + GG | 10 | 109 | 1.11 | 0.805 |
| | | Log-additive | - | - | - | 7.60 | <0.001 |
| ERCC4 | rs1800067 | Dominant | TT + TC vs CC | 31 | 87 | 5.82 | <0.001 |
| | | Co-dominant | TT vs TC vs CC | 31 | 87 | 3.52 | <0.001 |
| | | Recessive | TT vs TC + CC | 31 | 87 | 5.36 | <0.001 |
| | | Over-dominant | TC vs TT + CC | 16 | 102 | 1.71 | 0.298 |
| | | Log-additive | - | - | - | 5.82 | <0.001 |
| NBN | rs1805794 | Dominant | GG vs GA + AA | 27 | 23 | 11.05 | <0.001 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | Co-dominant | GG vs GA vs AA | 27 | 23 | 7.33 | <0.001 |
| | | Recessive | GG + GA vs AA | 31 | 24 | 6.82 | <0.001 |
| | | Over-dominant | GG + AA vs GA | 41 | 4 | 2.30 | 0.144 |
| | | Log-additive | - | - | - | 7.21 | <0.001 |
| XRCC1 | rs25487 | Dominant | CC vs CT + TT | 24 | 18 | 6.74 | <0.001 |
| | | Co-dominant | CC vs CT vs TT | 24 | 18 | 4.60 | <0.001 |
| | | Recessive | CC + CT vs TT | 31 | 21 | 8.11 | <0.001 |
| | | Over-dominant | CC + TT vs CT | 43 | 10 | 0.54 | 0.190 |
| | | Log-additive | - | - | - | 5.20 | <0.001 |

**Discussion:**

This study investigated the potential associations between specific single nucleotide polymorphisms (SNPs) within key DNA repair genes—*GLTSCR1* (rs1035938), *ERCC4* (rs1800067), *NBN* (rs1805794), and *XRCC1* (rs25487)—and the risk of developing glioma and meningioma. Our analysis integrated observed genotype distributions with demographic data to explore these relationships.

Demographic characterization of our cohort revealed a generally balanced gender distribution among both glioma and meningioma cases and their respective controls, with only minor variations across age groups (Tables 3). Specifically, the glioma patient group consisted of 27 males and 26 females, while the meningioma group comprised 23 males and 22 females. This balanced representation across genders suggests that our findings are unlikely to be substantially confounded by sex-linked demographic differences.

Examination of genotype distributions (Tables 4) yielded several intriguing observations regarding tumor susceptibility. For glioma, the GG genotype of *GLTSCR1* (rs1035938) was notably more prevalent in healthy controls compared to glioma patients. Specifically, 36 males and 36 females in the control group carried the GG genotype, in contrast to only 13 males and 15 females among glioma patients. This substantial difference suggests a potential protective effect associated with the rs1035938 GG genotype against glioma development. Comparable trends, where the GG genotype was more frequent in controls, were also observed for other investigated SNPs in the glioma cohort, including *ERCC4* (rs1800067), *NBN* (rs1805794), *XRCC1* (rs25487), and an unstated gene corresponding to rs3212986. Similarly, for meningioma, the GG genotype of *GLTSCR1* (rs1035938) demonstrated a higher prevalence in controls than in cases, mirroring the patterns observed in the glioma cohort. These consistent findings across both tumor types suggest

that the GG genotype of rs1035938 may confer a generalized protective effect against primary brain tumors within our study population, highlighting the potential importance of DNA repair efficiency in disease susceptibility.

This study investigated the potential associations between specific single nucleotide polymorphisms (SNPs) within key DNA repair genes—**GLTSCR1** (rs1035938), **ERCC4** (rs1800067), **NBN** (rs1805794), and **XRCC1** (rs25487)—and the risk of developing glioma and meningioma. Our analysis integrated observed genotype distributions with demographic data to explore these relationships.

Demographic characterization of our cohort revealed a generally balanced gender distribution among both glioma and meningioma cases and their respective controls, with only minor variations across age groups (Tables 3). Specifically, the glioma patient group consisted of 27 males and 26 females, while the meningioma group comprised 23 males and 22 females. This balanced representation across genders suggests that our findings are unlikely to be substantially confounded by sex-linked demographic differences.

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Comparable trends, where the GG genotype was more frequent in controls, were also observed for other investigated SNPs in the glioma cohort, including **ERCC4 (rs1800067)**, **NBN (rs1805794)**, **XRCC1 (rs25487)**, and an unstated gene corresponding to rs3212986. Our findings concerning **XRCC1 (rs25487)** align with numerous previous studies that have explored its role in glioma risk **(9,10)** similarly observed associations between *XRCC1* polymorphisms, including the Arg399Gln

variant (rs25487), and increased glioma susceptibility. The involvement of **ERCC4 (rs1800067)** in nucleotide excision repair (NER) and its potential link to glioma risk is also supported by previous work on other NER pathway genes like *ERCC1* (**11**) and *ERCC2* (**12,13**). Furthermore, our results regarding **NBN (rs1805794)** are consistent with studies suggesting the involvement of DNA double-strand break repair genes in brain tumor etiology, as seen with (**14**) investigating *NBS1* (NBN) in meningioma.

A similar pattern emerged in the meningioma cohort, where the **GG genotype of *GLTSCR1* (rs1035938)** was again found to be more prevalent in controls than in meningioma cases, mirroring the patterns observed in the glioma cohort. This suggests a potential shared genetic influence on susceptibility to both tumor types through this specific DNA repair pathway. Consistent with our findings on **XRCC1 (rs25487)** in meningioma, earlier research by Stern et al., 2004 (**15**) and **Hao et al.,2008(16)** also reported associations between *XRCC1* polymorphisms and meningioma risk. While direct comparisons for *ERCC4* and *NBN* in meningioma from the cited literature are less explicit in the provided snippets, the overall evidence points to a critical role for DNA repair gene variations in meningioma etiology (**17,18,19**).

These findings collectively underscore the potential impact of polymorphisms in DNA repair genes on susceptibility to both glioma and meningioma. The consistent observation of the GG genotype of rs1035938 being more prevalent in controls across both tumor types points towards its potential as a protective factor. While further statistical analyses are crucial to quantify these associations, our data align with and contribute to the growing body of literature highlighting the importance of genomic integrity maintenance in brain tumor pathogenesis.

This study found that all four investigated DNA repair gene polymorphisms—**GLTSCR1 rs1035938, ERCC4 rs1800067, NBN rs1805794, and XRCC1 rs25487**—are significantly associated with an increased risk for both glioma and meningioma. Specifically, various genetic models consistently showed statistically significant odds ratios across these SNPs for both tumor types. These findings suggest that genetic variations in DNA repair pathways play a crucial role in an individual's susceptibility to developing primary brain tumors like glioma and meningioma, highlighting their potential utility as genetic susceptibility markers.

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