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ISSN: 2684-1614
Journal of Tumour Research & Reports

Opinion Article

Pathophysiology and Molecular Characteristics of Meningioma

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DESCRIPTION

Meningiomas are tumours that develop from the meninges, particularly from the arachnoid cap cells that line the protective layers surrounding the brain and spinal cord. These specialised cells, under normal circumstances, maintain the structural integrity of the meninges, but under certain genetic and environmental influences, they can undergo abnormal proliferation. This unchecked growth results in the formation of discrete masses that may exert pressure on adjacent neural tissue, producing neurological symptoms. The majority of meningiomas are slow-growing and benign, yet variations in cellular characteristics and molecular profiles can lead to more aggressive forms that carry higher risks of recurrence. Genetic alterations are a central aspect of meningioma development. One of the most frequently observed changes involves deletions on chromosome 22, which affect the Neuro Fibromatosis type 2 (NF2) gene. NF2 encodes a protein called merlin that regulates cell growth and maintains contact inhibition, preventing excessive proliferation. Loss of merlin function allows arachnoid cap cells to divide uncontrollably, initiating tumour formation. Beyond NF2, additional chromosomal changes, including mutations in chromosomes 1, 14 and 9, have been linked to more atypical or aggressive behaviour, highlighting the role of multiple genetic factors in tumour progression.

Histological examination provides insights into the cellular architecture of meningiomas and helps classify them into subtypes such as meningothelial, fibroblastic, transitional and psammomatous forms. These subtypes display distinct arrangements of spindle or polygonal cells, as well as characteristic patterns like whorls and psammoma bodies. Higher-grade tumours, identified as atypical or anaplastic, exhibit increased mitotic activity, nuclear atypia and local invasion into surrounding tissue. These features are closely associated with rapid growth and a higher likelihood of recurrence, emphasizing the importance of detailed pathological evaluation for prognosis and treatment planning. Molecular signalling pathways play a critical role in sustaining tumour growth and survival. Aberrant activation of growth factor receptors, including platelet-derived growth factor and epidermal growth factor receptors, promotes cell proliferation and angiogenesis, supporting tumour expansion. Dysregulation of intracellular pathways such as (PI3K/AKT/mTOR and MAPK) further enhances survival of meningioma cells by preventing apoptosis and enabling adaptation to local environmental conditions. Epigenetic modifications, including Deoxyribonucleic Acid (DNA) methylation and histone acetylation, have been identified as additional mechanisms influencing gene expression and promoting tumorigenesis.

Hormonal factors are also implicated in meningioma biology. Many tumours express progesterone receptors, with a smaller proportion expressing estrogen receptors, which may contribute to the higher incidence observed in women. Clinical observations suggest that tumours can enlarge during periods of hormonal fluctuation, such as pregnancy or hormone therapy. The precise mechanisms by which these hormonal signals stimulate growth remain under investigation, but receptor expression patterns provide useful information for understanding tumour behaviour and potential therapeutic considerations. Environmental influences, particularly exposure to ionizing radiation, have been associated with increased risk of meningioma development. Radiation can induce DNA damage and chromosomal instability in arachnoid cap cells, contributing to neoplastic transformation. Inherited genetic syndromes, though rare, can predispose individuals to multiple tumours through germline mutations affecting tumour suppressor genes. The interplay between genetic susceptibility, environmental exposure and spontaneous mutations contributes to the multifactorial nature of meningioma pathophysiology. The location of the tumour significantly influences its clinical impact. Tumours adjacent to cranial nerves, the optic pathway or critical brain regions may produce symptoms even when relatively small, while those in non-eloquent regions may remain undetected until they achieve substantial size. This anatomical consideration, combined with molecular and histological features, informs treatment decisions, surgical planning and prognosis assessment.

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Received: 17-Nov-2025, Manuscript No. JTRR-25-30697; **Editor assigned:** 20-Nov-2025, Pre QC No. JTRR-25-30697(PQ); **Reviewed:** 4-Dec-2025, QC No. JTRR-25-30697; **Revised:** 11-Dec-2025, Manuscript No. JTRR-25-30697 (R); **Published:** 18-Dec-2025, DOI: 10.35248/2684-1614.25.10.279

Citation: Traven LR (2025). Pathophysiology and Molecular Characteristics of Meningioma. J Tum Res Reports.10:279.

In summary, meningiomas result from complex interactions among genetic alterations, disrupted signaling pathways, hormonal influences and environmental factors. Detailed understanding of histological and molecular characteristics allows clinicians to classify tumours, anticipate behaviour and plan management strategies effectively. Integrating insights from cellular biology, molecular pathways and anatomical considerations enhances diagnostic precision and supports informed clinical decision-making for patients affected by meningioma.