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Morphology Matters: A Multidimensional Perspective on Cancer Cell Biology

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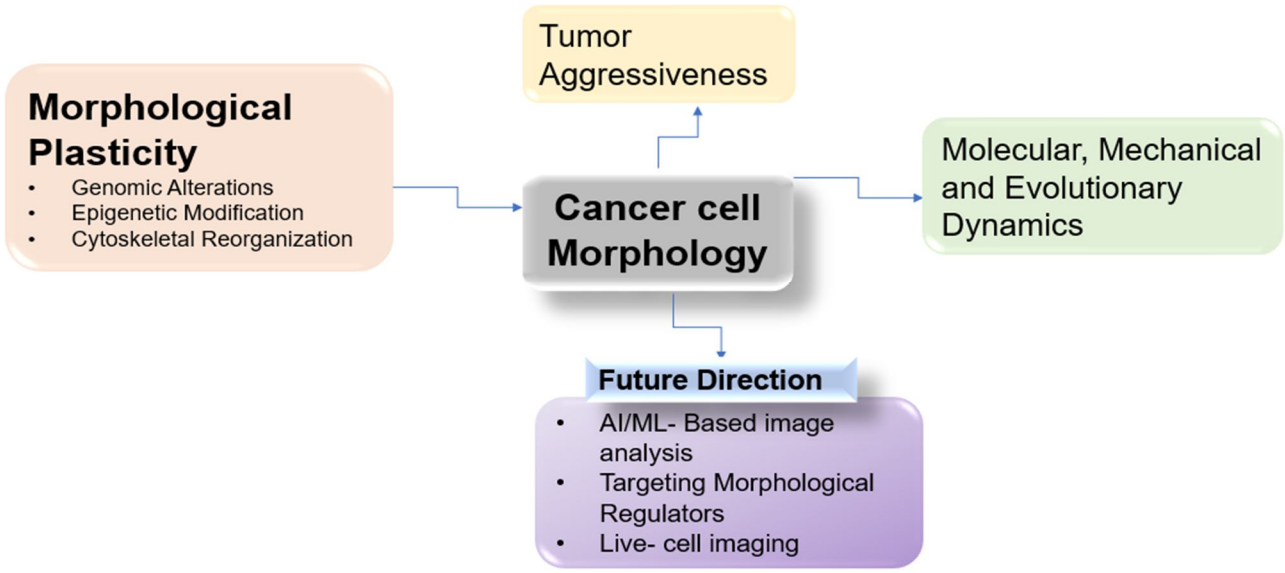
Abstract: ***Cancer cell morphology reflects the dynamic interplay between genetic, epigenetic, and environmental factors that drive tumor progression. This review explores the morphological evolution of cancer cells as a phenotypic manifestation of underlying molecular dynamics, drawing on recent advances in evolutionary biology, cytoskeletal remodeling, computational pathology, and tumor metabolism. Clonal evolution, both gradual and punctuated, generates morphological diversity that is shaped by selective pressures from the tumor microenvironment and therapeutic interventions. Studies highlights the structural and mechanical properties, such as reduced cellular stiffness and altered actin organization, are linked to metastatic potential and cytoskeletal deregulation. Oncogenic signaling pathways (e.g., PI3K/AKT, Ras/MAPK) and metabolic reprogramming further modulate cell shape and behavior. High-throughput imaging and machine learning have enabled quantification of both static and dynamic morphological traits, correlating them with chromosomal instability and clinical outcomes. Morphological heterogeneity has emerged as a powerful diagnostic and prognostic biomarker, with deep learning-derived metrics providing scalable tools for cancer classification and risk stratification. Collectively, these insights underscore the clinical utility of integrating morphological analysis with molecular profiling, offering new directions for personalized cancer diagnosis and therapy.***

Keywords: Clonal evolution; Tumor heterogeneity; Cytoskeletal remodeling; Epithelial-mesenchymal transition (EMT); Morphometric analysis; Tumor microenvironment.

GRAPHICAL ABSTRACT



I. INTRODUCTION

Understanding cancer cell morphology is crucial in advancing cancer research and developing effective treatment strategies, as it reflects intricate molecular and evolutionary changes during tumorigenesis and progression. As tumors evolve, cells undergo progressive morphological transformations ranging from loss of polarity and cytoskeletal reorganization to nuclear atypia and increased deformability driven by a combination of genetic, epigenetic, and mechanical alterations.

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These changes are initiated through stepwise mutations in proto-oncogenes and tumor suppressor genes such as *RAS*, *TP53*, *MYC*, and *RB*, which disrupt growth regulation, apoptosis, and cellular architecture [1], [2]. Classical Darwinian models of cancer evolution suggest that these genetic mutations accumulate gradually under selective pressures, giving rise to subclonal diversification and morphological heterogeneity within tumors [3]. However, recent findings suggest that non-Darwinian mechanisms, including neutral evolution, chromosomal catastrophes (e.g., chromothripsis, chromoplexy), and epigenetic shifts like the CpG island methylator phenotype (CIMP), also contribute significantly to both phenotypic diversity and disease trajectory [1], [4]. Simultaneously, cancer cells remodel their cytoskeletal components actin filaments, microtubules, and intermediate filaments which influence shape, mechanical stiffness, and motility. These biomechanical alterations facilitate transitions between different migration modes (mesenchymal, amoeboid, and collective) and are regulated by signaling pathways such as Rho/ROCK, Rac1, and YAP/TAZ [5] Mechanical inputs from the tumor microenvironment, such as ECM stiffness and interstitial flow, activate mechanotransduction cascades that feed into transcriptional programs governing morphology and invasion. [5] Importantly, nuclear morphology characterized by envelope irregularity, chromatin reorganization, and altered Lamin composition undergoes significant changes in aggressive cancers, facilitating migration through confined spaces and influencing gene expression [5], [6]. These insights collectively indicate that morphological evolution in cancer cells is a complex outcome shaped by somatic mutation, selective pressures, epigenetic remodeling, and biomechanical forces, underscoring the multifaceted nature of this process. Understanding the interplay between form and function in cancer not only enhances the biological understanding of malignancy but also opens new avenues for therapeutic targeting of morphological regulators and diagnostic exploitation of phenotypic plasticity [4], [5], [6]. Cancer cell evolution is a multidimensional process that encompasses genetic mutations, selection, profound alterations in metabolism, intercellular cooperation, and morphological plasticity. Otto Warburg's seminal observations in the 1956s established that cancer cells prefer aerobic glycolysis over oxidative phosphorylation even in the presence of oxygen, a phenomenon later termed the "Warburg effect," suggesting that impaired mitochondrial respiration may be a primary driver of malignant transformation, highlighting the critical role of metabolic reprogramming in cancer development [6]. Building on this foundation, contemporary studies have shown that metabolic reprogramming such as enhanced glycolysis, mitochondrial remodeling, and lipid biosynthesis is essential for supporting unrestrained proliferation and biosynthesis during tumor growth, highlighting its pivotal role in cancer progression. This metabolic shift enables cancer cells to meet the demands of anabolic growth and redox balance, driven by the activation of oncogenes such as MYC and PI3K, as well as the loss of tumor suppressors like p53, FH, and SDH, leading to a state of metabolically convergent evolution [6], [7]. In parallel, the tumor microenvironment fosters not only competition but also cooperation among heterogeneous tumor subclones, as highlighted by Pienta et al., who proposed that tumor cells can share diffusible growth factors and metabolic by-products, enhancing survival and promoting malignant progression through by-product mutualism [8]. Morphologically, these evolving cancer cells display increasing heterogeneity, including variations in size, polarity loss, nuclear abnormalities, and migratory plasticity [9]. Moreover, cancer therapies can paradoxically induce senescence a state of permanent proliferation arrest characterized by enlarged, polyploid morphology and sustained secretory activity (SASP) which, while initially tumor-suppressive, may contribute to recurrence and therapy resistance if senescent cells re-enter the cell cycle or support the tumor microenvironment through inflammatory signaling, highlighting the potential challenges in managing induced senescence in cancer treatment [10]. Together, these findings illustrate that cancer is not a static genetic disorder but a dynamic, adaptive system intricately shaped by energy metabolism, ecological interactions, and stress responses, underscoring the complexity of cancer biology. This review aims to synthesize current knowledge on the morphological evolution of cancer cells, focusing on the impact of signaling pathways, metabolic changes, epigenetic modifications, and cell-cell interactions on cell morphology and function. By mapping how morphology reflects oncogenic signaling, mechanical inputs, metabolic demands, and evolutionary pressures, we argue for the inclusion of morphological plasticity as a central axis in both cancer biology research and therapeutic innovation. This perspective may help identify new biomarkers of progression and resistance, and pave the way for novel interventions that target not only genetic mutations but also the form and behavior of malignant cells in context.

II. EVOLUTIONARY BASIS OF CANCER CELL MORPHOLOGY

Peter Nowell's seminal concept of clonal evolution, further expanded by Greaves and Maley, describes tumors as evolving populations subject to natural selection, where genetic diversity fuels subclonal competition and morphological diversification [11]. Cancer evolves through successive genetic alterations, many of which confer fitness advantages that allow subclones to expand under selective pressures such as therapy, immune response, or nutrient deprivation. These alterations do not occur in a vacuum; they dynamically interact with each other and with the tumor microenvironment, producing phenotypic outputs including changes in cellular morphology.

While many changes accumulate gradually over time through point mutations and small indels, some transformations are more dramatic [3] emphasized that macroevolutionary events like chromothripsis (massive chromosomal shattering and rearrangement) or whole-genome doubling can act as evolutionary leaps, generating sudden morphological shifts. Such punctuated alterations can produce novel cellular behaviors and morphologies that would be improbable through incremental evolution alone [3]. Furthermore, parallel evolution where different subclones acquire distinct mutations in the same pathway can result in convergent morphological phenotypes across diverse genomic backgrounds. The tumor microenvironment (TME), as described by [12], exerts profound influence on the evolution of cancer morphology. The TME is composed of non-cancerous cells (e.g., fibroblasts, immune cells), extracellular matrix components, and gradients of oxygen, pH, and nutrients. These elements impose both mechanical and chemical pressures on tumor cells. One of the most morphologically transformative responses to the TME is epithelial-mesenchymal transition (EMT), in which epithelial cells lose their polarity and adhesion properties, adopting a spindle-like, migratory morphology associated with increased invasiveness and metastatic potential [13].

III. MOLECULAR DRIVERS OF MORPHOLOGICAL CHANGE

Cancer cell morphology is largely governed by the cytoskeleton, an internal scaffold composed of actin filaments, microtubules, and intermediate filaments. Structural alterations in cytoskeletal proteins or their regulators can significantly affect cell shape and mechanics. Lekka et al. used atomic force microscopy to measure mechanical stiffness in cancer cells and found that malignant and particularly metastatic cells exhibit reduced stiffness compared to normal cells. This biomechanical softening, linked to cytoskeletal disorganization (especially actin filament remodeling), facilitates cellular deformation, enhancing motility and invasiveness. Multiple signaling pathways implicated in oncogenesis also regulate morphology. The PI3K/AKT/mTOR pathway, for instance, controls not only growth and survival but also impacts actin cytoskeletal dynamics through downstream effectors. Similarly, Ras/MAPK signaling promotes filopodia and lamellipodia formation, driving cell spreading and migration [14]. In Another study it is demonstrated that these pathways often converge on metabolic reprogramming, such as the Warburg effect, which supports biosynthetic demands for membrane and cytoskeletal components required for morphological remodeling. Transcription factors like YAP/TAZ and HIF1α further mediate morphology-related gene expression programs in response to environmental cues. Epigenetic mechanisms, including DNA methylation and histone modifications, regulate genes associated with the cytoskeleton, cell adhesion, and motility. Single-cell transcriptomic analyses provide insight into how transcriptional programs correspond with specific morphological states [15]. In silico study developed a computational tool (TISMorph) to quantify cellular shape and actin architecture from images of various cancer cell lines. Their results showed that even cells with the same genetic background but differing metastatic potential exhibited distinct morphological signatures. This underscores the tight coupling between morphology and dynamic transcriptional landscapes [9]. Highlighted variability in nuclear size, shape, chromatin distribution, and nucleolar structure in cancer cells. However, no consistent ultrastructural markers were found to universally distinguish malignant nuclei. Many observed nuclear abnormalities could result from secondary effects (e.g., hypoxia, necrosis) rather than primary malignancy features [16]. Digital morphometric analysis of glioblastoma nuclei revealed that nuclear shape and spatial relationships (e.g., distance between nuclei) significantly correlate with patient survival. These morphological traits had prognostic value independent of age or surgical resection status [17]. Cancer cells are generally softer and more deformable than normal cells, regardless of whether they originate from the same or different organs. This increased deformability correlates with disease progression and plays a critical role in facilitating metastasis by enabling cancer cells to migrate more easily through tight extracellular matrix (ECM) spaces. Interestingly, while tumors as a whole often feel stiff due to the surrounding stroma and tissue architecture, individual cancer cells within them exhibit greater softness. This paradox is attributed to cytoskeletal remodeling, alterations in nuclear structure, and adaptive responses to the tumor microenvironment, all of which contribute to the mechanical flexibility that supports cancer invasiveness and dissemination. Cell mechanics vary with culture conditions and measurement techniques. Use of techniques like atomic force microscopy (AFM), optical stretching, and micropatterning has revealed that local morphology (such as nucleus size and cell spread area) can significantly alter measurement outcomes, suggesting that standardized conditions are essential for reproducible biomechanical assessments [18]. Cancer cells exhibit altered cell-cell adhesion and more convoluted membrane surfaces compared to normal epithelial cells. These changes result in fewer and morphologically distinct intercellular contacts, which likely contribute to the increased motility and invasiveness observed in malignant cells [19]. Supporting this, carbon replica studies have revealed that cancer cell membranes are notably rougher than those of normal cells, reflecting structural adaptations that may facilitate detachment, migration, and invasion through surrounding tissues [20]. During epithelial-to-mesenchymal transition (EMT), cancer cells undergo significant structural remodeling, including altered expression of intermediate filaments such as keratins and vimentin, which directly impacts cellular stiffness and deformability.

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These cytoskeletal changes are accompanied by nuclear alterations, where cancer cell nuclei often become larger, more irregular, and exhibit modified lamin expression. Such nuclear remodeling contributes to reduced nuclear stiffness, making it easier for cancer cells to squeeze through confined spaces, thereby facilitating metastasis. Nuclear envelope rupture and dynamic remodeling further enhance this invasive potential [18], [21]. In parallel, cancer cells reprogram their metabolic pathways most notably through the Warburg effect to meet the high biosynthetic demands of rapid proliferation. This metabolic shift not only fuels growth but also indirectly influences cell morphology and mechanics. The accumulation of metabolic intermediates and adaptation to stress conditions like hypoxia or nutrient scarcity lead to changes in cell volume, reorganization of the cytoskeleton, and alterations in nuclear architecture [6]. Together, these metabolic and structural adaptations provide cancer cells with enhanced plasticity and resilience in hostile tumor microenvironments.

Table 1. Summary of key molecular, mechanical, and analytical factors influencing cancer cell morphology

Category	Key Findings
Cytoskeleton Remodeling	Actin changes increase motility
Cell Mechanics	Softer cells migrate easier
PI3K/AKT/mTOR Pathway	Shapes cytoskeleton and growth
Ras/MAPK Signaling	Drives spreading, filopodia, lamellipodia
Warburg Effect	Fuels shape, biosynthesis changes
YAP/TAZ, HIF1α	Regulate shape-related genes
Epigenetics	Alters cytoskeleton, motility genes
TISMorph (In Silico Tool)	Identifies distinct metastatic morphologies
Single-Cell Transcriptomics	Morphology matches gene expression
Nuclear Variability	Irregular nuclei, unclear malignancy markers
Glioblastoma Morphometry	Nuclear shape predicts survival
Measurement Variability	Mechanics shift with technique, conditions
Adhesion & Membranes	Weak contacts, rough surfaces
EMT Remodeling	Alters stiffness, nucleus, invasiveness
Nuclear Envelope Dynamics	Remodeling aids squeezing, migration
Tumor Paradox	Cells soft; tumor feels stiff
Metabolic Adaptation	Stress reshapes cell, nucleus

IV. QUANTIFICATION OF MORPHOLOGICAL DIVERSITY

Traditionally, pathologists use morphological characteristics like nuclear pleomorphism, mitotic rate, and gland formation to grade tumors. However, quantitative digital pathology is revolutionizing this approach. Sali et al. analyzed over 68 million H&E-stained cancer cells using deep learning algorithms to generate a cancer cell diversity score. They found that higher morphological diversity within a tumor was strongly associated with chromosomal instability and poor clinical outcomes. Importantly, this score was prognostic across multiple cancer types and added value beyond existing clinical factors. Static images provide only a snapshot of cancer cell morphology. Dynamic imaging allows for analysis of real-time behaviors such as migration, division, and protrusion dynamics [22]. In a study with time-lapse microscopy of glioblastoma and astrocyte cells on aptamer-functionalized surfaces. Using machine learning algorithms (SVM, Random Forest, Naive Bayes), they were able to classify cancerous vs. non-cancerous cells with over 85% accuracy based on dynamic features. This approach highlights the diagnostic potential of analyzing cell behavior, not just structure. Modern computational tools allow for high-throughput, multidimensional analysis of cell morphology [23]. Alizadeh et al. and Sali et al. have shown that deep learning models can extract latent morphological features from image datasets that are not visible to the human eye but correlate strongly with molecular profiles and clinical outcomes. Such tools not only enhance objectivity and scalability in cancer diagnostics but also serve as bridges linking molecular dynamics with phenotypic expression.

V. CLINICAL IMPLICATIONS

Histopathological evaluation remains a gold standard in cancer diagnostics. The integration of computational image analysis now enables automated, standardized assessment of features such as nuclear size, shape, and texture. Sali et al.'s cancer cell diversity score is a prime example of how morphological information can be quantified and used as a biomarker. Such methods promise to reduce inter-observer variability and extend access to expert-level diagnosis globally.

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Morphological heterogeneity is not just a descriptive feature but a functional indicator of tumor aggressiveness and adaptability. Sali et al. demonstrated that tumors with higher morphological diversity tend to harbor more chromosomal instability, a hallmark of aggressive disease. Thus, morphology can serve as a surrogate for underlying genomic chaos and a predictor of poor outcomes. Integrating morphology with genomic and transcriptomic data allows for multidimensional risk stratification. Morphological features can inform therapeutic targeting. For example, tumors enriched in mesenchymal-like cells may benefit from therapies targeting EMT pathways, such as TGF-β or Wnt inhibitors[13], [24]. Similarly, identification of drug-resistant morphological states through live-cell imaging or computational models could guide adaptive therapy strategies. Ultimately, combining morphologic insights with molecular profiling provides a richer, more actionable understanding of tumor biology.

VI. OBSERVATION

The collective findings from the reviewed studies reveal a strong and consistent association between cancer cell morphology and the underlying molecular, mechanical, and evolutionary dynamics. Morphological plasticity manifested in features such as nuclear atypia, cytoskeletal reorganization, and increased deformability closely reflects genomic and epigenetic alterations, including mutations in key oncogenes and tumor suppressors (e.g., TP53, RAS, MYC), as well as large-scale events like chromothripsis. These molecular changes, along with signaling through pathways such as YAP/TAZ, PI3K/AKT, and Rho/ROCK, dynamically reshape the cytoskeleton and influence cancer cell behavior, promoting invasiveness and adaptability. Moreover, morphological heterogeneity itself emerges as a functional indicator of tumor aggressiveness and poor prognosis. Quantitative studies employing deep learning and high-throughput image analysis demonstrate that increased morphological diversity correlates strongly with chromosomal instability and unfavourable clinical outcomes. These findings collectively underscore that morphology is not merely a passive phenotype but a biologically informative and predictive dimension of cancer progression.

VII. CONCLUSION

The integrated analysis of cancer cell morphology across genetic, mechanical, metabolic, and ecological contexts reveals that morphological evolution is not a secondary feature of cancer but a central axis of malignant progression. Morphology embodies the cumulative impact of oncogenic signaling, metabolic reprogramming, microenvironmental pressures, and evolutionary selection. It serves as both a mirror and mediator of cancer dynamics, offering valuable insights into tumor behavior, adaptability, and treatment response. The reviewed literature affirms that morphological plasticity is a key phenotypic expression of cancer's evolutionary trajectory, and thus, should be incorporated as a core dimension in cancer diagnostics, prognostics, and therapy development. Embracing this perspective can catalyze the discovery of novel morphological biomarkers and the development of targeted interventions that go beyond genomic alterations to include the structural and mechanical vulnerabilities of malignant cells.

VIII. FUTURE DIRECTION

Future research should focus on integrating cancer cell morphology with multi-omic data to uncover links between molecular alterations and phenotypic states. Advancements in live-cell imaging and mechanobiology can reveal how cells dynamically adapt under treatment. Expanding the use of AI-driven image analysis will enhance diagnostics and prognosis prediction. Additionally, targeting morphological regulators such as cytoskeletal components and mechanotransduction pathways may offer novel therapeutic strategies. Emphasizing morphology as a functional hallmark of cancer holds promise for improving both understanding and treatment of malignancies.

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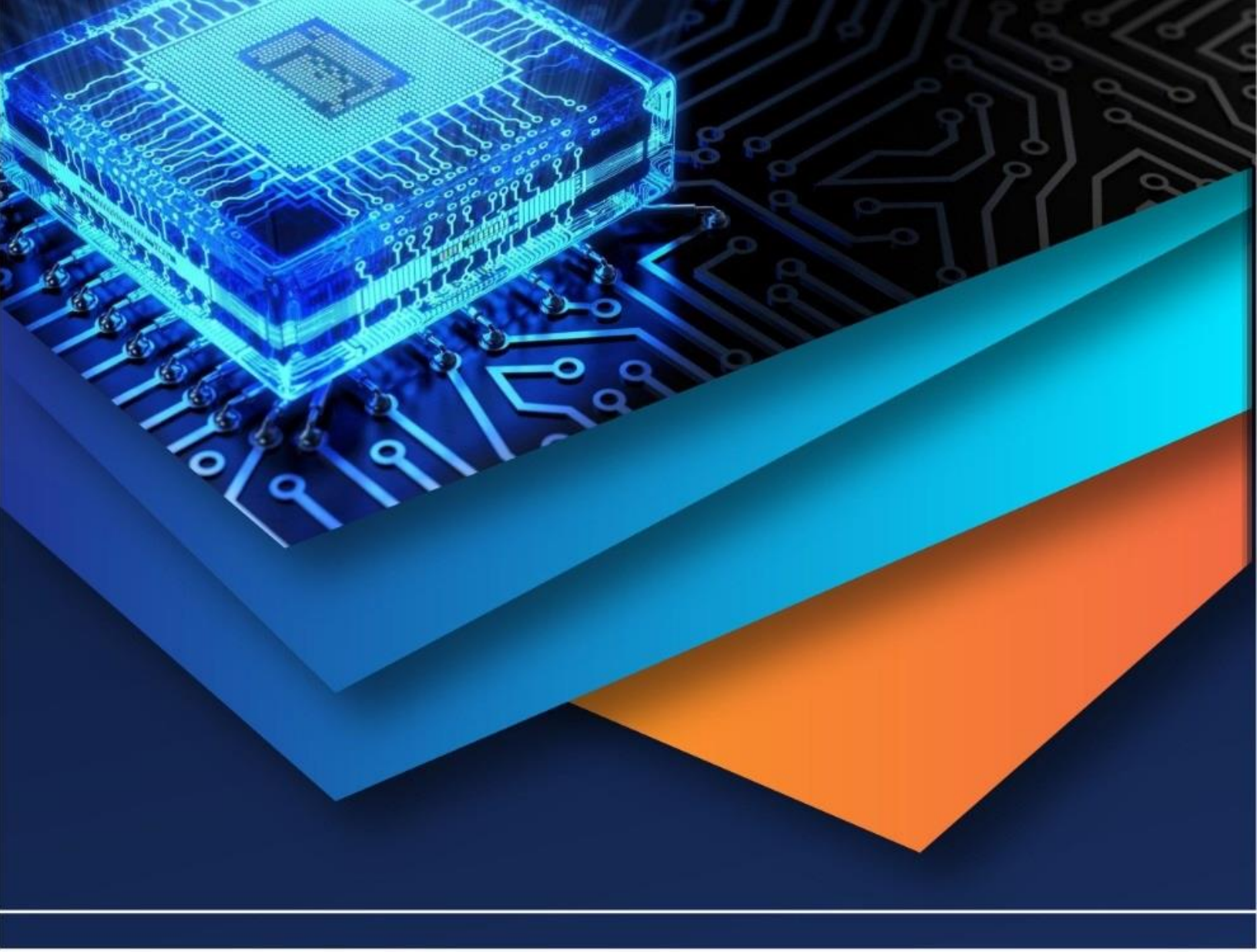

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